

Psychology Units 3 & 4

Chapter 4 — Stress as a psychobiological process

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Chapter 4 Stress as a psychobiological process — 4A Stressors and the acute and chronic stress response

Theory

Stress is a state of physiological and psychological arousal that occurs when a person appraises that the demands of a situation exceed, or tax, their ability to cope. A **stressor** is any event, object or circumstance — internal or external — that **produces a stress response**. The same stressor can be experienced very differently by different people, but once a stress response begins it involves the whole person: the **body** and the **mind**.

Internal and external stressors. Stressors are classified by where they come from.

- An **external stressor** originates **outside** the person, in their environment. Examples include an upcoming exam, a demanding job, financial pressure, a car narrowly missing you, conflict with a friend, a crowded train, or extreme heat.
- An **internal stressor** originates **inside** the person — in their own thoughts, feelings, attitudes or physiological state. Examples include worrying or ruminating about the future, pessimistic or catastrophic thinking, perfectionism, low self-esteem, physical illness, hunger or pain.

The *same situation* often involves both: sitting an exam is an **external** stressor, while the thought “I am going to fail” is an **internal** stressor. Both kinds can trigger the same stress responses.

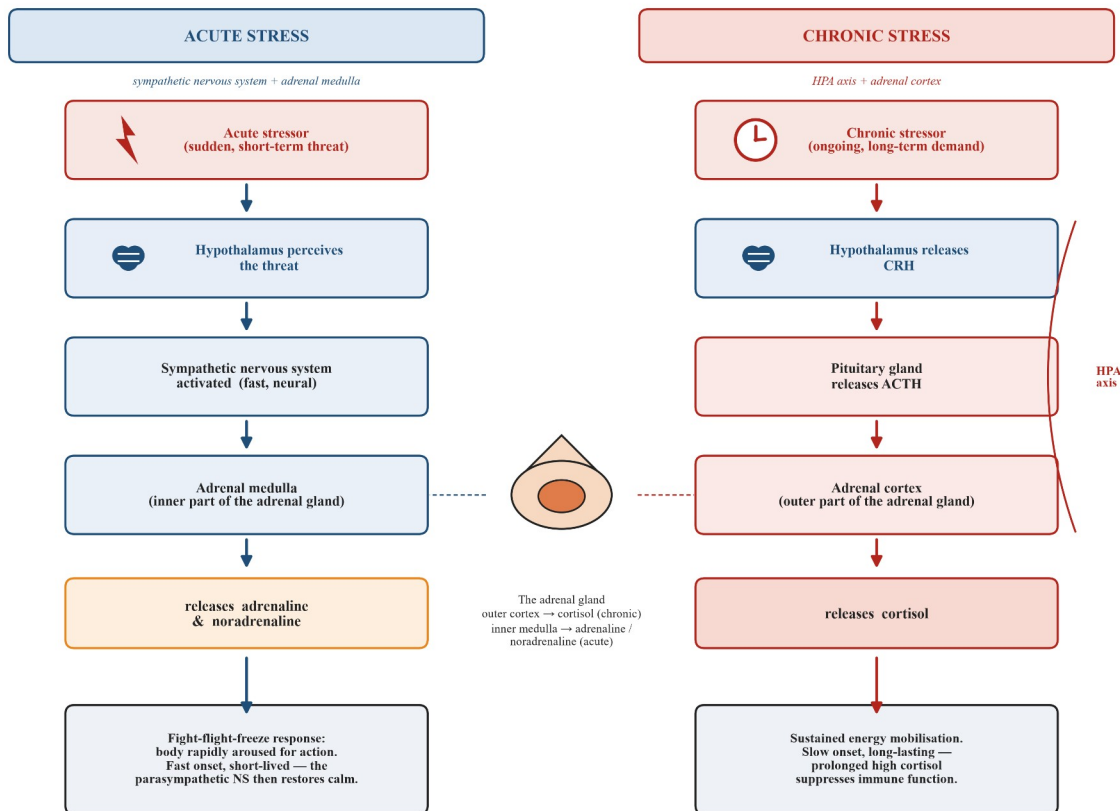
Psychological and physiological stress responses. A stress response has two components.

- A **physiological stress response** is the body’s automatic **biological** reaction — for example an increased heart rate, faster breathing, sweating, muscle tension, the release of stress hormones (adrenaline, noradrenaline and cortisol), a suppressed immune system, fatigue and disrupted sleep or digestion. These are **bodily** changes.
- A **psychological stress response** is the person’s **cognitive, emotional and behavioural** reaction — for example feeling anxious, irritable or overwhelmed (emotional), racing thoughts or difficulty concentrating (cognitive), and changes in behaviour such as withdrawing socially, snapping at the people around them or putting off tasks (behavioural). These are **mental and behavioural** changes.

Acute versus chronic stress. The stress response unfolds differently depending on how long the stressor lasts.

- **Acute stress** is **short-term** stress produced by a **sudden**, immediate stressor (for example a near-miss in traffic). Its arousal is **intense but brief**.
- **Chronic stress** is **prolonged, ongoing** stress produced by a stressor that persists over a long period (for example months of financial hardship). Its arousal is **lower but sustained**.

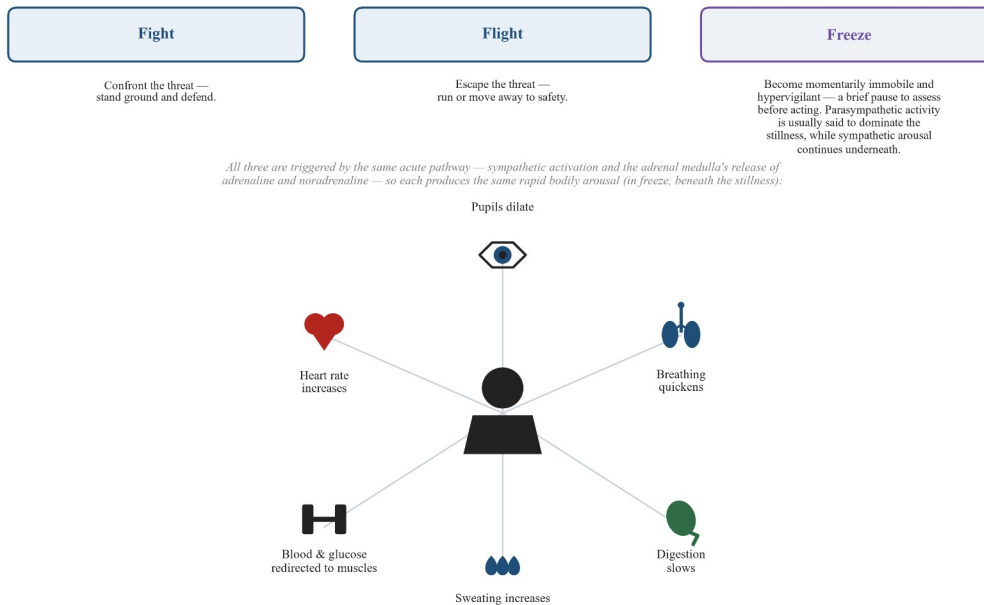
The two involve **different biological pathways**, and keeping them apart is essential.



The acute response — the fight-flight-freeze response. When the brain (the **hypothalamus**) perceives a sudden threat, it activates the **sympathetic nervous system** — the branch of the autonomic nervous system that arouses the body for action. The sympathetic nervous system stimulates the **adrenal medulla** (the **inner** part of the adrenal glands, which sit above the kidneys), which releases the hormones **adrenaline** and **noradrenaline** into the bloodstream. Because this pathway is largely **neural**, it acts within **seconds**. Adrenaline and noradrenaline, together with direct sympathetic activation, produce rapid bodily arousal that prepares the person to deal with the threat in one of three ways:

- **Fight** — confront the threat (stand ground and defend).
- **Flight** — escape the threat (run or move away to safety).
- **Freeze** — become momentarily **immobile and hypervigilant**. The freeze response is commonly described as a brief, **parasympathetic-dominated** state of tense stillness in which the person stops and “keeps still” to assess the threat, while remaining physiologically aroused and ready to switch to fight or flight. It is adaptive when moving would draw a predator’s attention or when the best first step is to gather information.

The fight-flight-freeze response (acute stress)

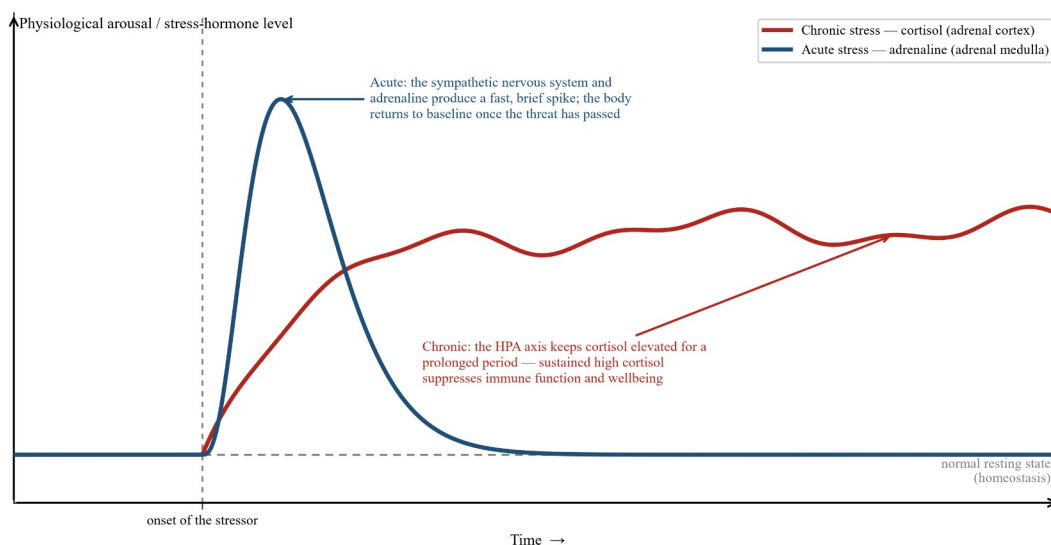


The physiological arousal of the fight-flight-freeze response includes an **increased heart rate**, **faster breathing**, **dilated pupils**, **increased blood flow and glucose to the skeletal muscles**, **increased sweating** and **slowed digestion** — all of which ready the body for sudden physical action. Acute stress is **self-limiting**: once the threat has passed, the **parasympathetic** nervous system restores the body toward its normal resting state (homeostasis).

The chronic response — the role of cortisol. When a stressor is **ongoing**, the body relies on a slower, hormonal pathway called the **HPA axis** (the Hypothalamic–Pituitary–Adrenal axis):

1. The **hypothalamus** releases a signalling hormone (corticotropin-releasing hormone, CRH).
2. This stimulates the **pituitary gland** to release adrenocorticotropic hormone (ACTH) into the bloodstream.
3. ACTH travels to the **adrenal cortex** (the **outer** part of the adrenal glands), which releases **cortisol**.

Cortisol is the body's main **chronic** stress hormone. In the short term it is **adaptive**: it increases the availability of energy (releasing glucose into the blood) so the body can keep meeting a sustained demand, and it dampens non-essential functions. Because it works through the bloodstream, cortisol takes **minutes** to have its effect but can keep the body mobilised for **hours, days or longer**. The problem is that **prolonged, elevated cortisol** — the hallmark of **chronic stress** — becomes harmful: it **suppresses the immune system** (leaving the person more vulnerable to illness) and is associated with fatigue, disrupted sleep and reduced wellbeing.



Key distinctions to keep straight.

- **Acute** = sympathetic nervous system + adrenal medulla → **adrenaline and noradrenaline** (fast, neural, brief). **Chronic** = HPA axis + adrenal cortex → **cortisol** (slower, hormonal, sustained). Do **not** conflate the two: the adrenal **medulla** releases **adrenaline/noradrenaline**, whereas the adrenal **cortex** releases **cortisol**.
- The **medulla** is the **inner** part of the adrenal gland; the **cortex** is the **outer** part.
- A **stressor** is the trigger; the **stress response** is the reaction. Every stress response has both a **physiological** (bodily) and a **psychological** (cognitive/emotional/behavioural) component.
- **Freeze** is a genuine third acute response — not “doing nothing”, but an active, alert immobility.

How to analyse a scenario. (1) Identify the **stressor** and classify it as **internal** (from the person’s own thoughts/body) or **external** (from the environment). (2) Decide whether the stress described is **acute** (sudden, brief) or **chronic** (ongoing, prolonged). (3) Map the correct pathway — for acute, **sympathetic NS → adrenal medulla → adrenaline/noradrenaline → fight, flight or freeze**; for chronic, **HPA axis → adrenal cortex → cortisol → sustained effects**. (4) Where asked, separate the **physiological** responses from the **psychological** responses.

Worked examples

Example 1 — scaffolded

Before a job interview tomorrow, Priya keeps thinking, “I will freeze up and embarrass myself.” Her heart is racing, and she feels anxious and cannot concentrate on anything else. Identify the internal stressor and the external stressor, and classify each of Priya’s responses as physiological or psychological.

Working	Comment
The interview tomorrow comes from Priya’s environment → external stressor .	An external stressor originates outside the person.
The thought “I will freeze up and embarrass myself” comes from Priya’s own mind → internal stressor .	An internal stressor originates in the person’s own thoughts/feelings.
Her racing heart is an automatic bodily change → physiological response .	Physiological responses are biological/bodily.
Feeling anxious and being unable to concentrate are emotional and cognitive reactions → psychological responses .	Psychological responses are cognitive, emotional or behavioural.

Example 2 — scaffolded

A cyclist is riding home when a car suddenly pulls out in front of her. Within a second she swerves hard and avoids a collision; her heart is pounding. Set out the biological pathway that produced this acute stress response, and classify it as acute or chronic.

Working	Comment
The car pulling out is a sudden, short-term threat → this is acute stress.	Acute stress = a sudden stressor producing brief, intense arousal.
The hypothalamus perceives the threat and activates the sympathetic nervous system .	The sympathetic branch of the autonomic nervous system arouses the body.
The sympathetic nervous system stimulates the adrenal medulla , which releases adrenaline and noradrenaline .	Name the inner part of the adrenal gland and the two hormones.
These hormones produce rapid arousal (heart pounding, blood to the muscles), enabling the flight response (swerving away).	This is the fight-flight-freeze response — here, flight.

Example 3 — unscaffolded

For the past four months, Marcus has been caring for an unwell parent while also working full time, and feels constantly under pressure. Explain the role of cortisol in his stress response, naming the pathway involved, and describe one consequence if his cortisol remains elevated.

Marcus is experiencing **chronic stress**, because the stressor (ongoing caring and work demands) is **prolonged** rather than sudden. Prolonged stress is managed by the **HPA axis**: his **hypothalamus** signals the **pituitary gland**, which releases a hormone that stimulates the **adrenal cortex** — the **outer** part of the adrenal glands — to release **cortisol**. Cortisol keeps energy (glucose) available so Marcus's body can keep meeting the sustained demand. However, because his cortisol stays **elevated over a long period**, it begins to **suppress his immune system**, leaving him more susceptible to illness and infection. ∴ cortisol, released via the HPA axis from the adrenal cortex, sustains Marcus's chronic stress response, but its prolonged elevation harms his immune function.

Example 4 — unscaffolded

Distinguish between the biological pathway of the acute stress response and that of the chronic stress response.

In the **acute** stress response, a sudden stressor activates the **sympathetic nervous system**, which stimulates the **adrenal medulla** (the inner adrenal gland) to release **adrenaline** and **noradrenaline**. Because this pathway is largely **neural**, it acts within **seconds**, producing an intense but **brief** burst of arousal (the fight-flight-freeze response) that subsides once the threat passes. In the **chronic** stress response, a prolonged stressor activates the **HPA axis** (hypothalamus → pituitary → adrenal cortex), and the **adrenal cortex** (the outer adrenal gland) releases **cortisol**. Because this pathway is **hormonal**, it is **slower** to act but keeps the body mobilised for a **sustained** period. ∴ acute stress works quickly through the sympathetic nervous system and adrenaline/noradrenaline from the adrenal medulla, whereas chronic stress works more slowly through the HPA axis and cortisol from the adrenal cortex.

Practice questions

Scaffolded

Q1. Define each of the following: (a) a stressor; (b) an internal stressor; (c) an external stressor.

Q2. Since losing his job, Sam has been worrying every night about money (an ongoing concern). He often feels hopeless, has trouble sleeping and frequent tension headaches, and has started avoiding his friends. (a) Identify the external stressor and the internal stressor. (b) Identify one **psychological** stress response. (c) Identify one **physiological** stress response.

Q3. A hiker rounds a bend and comes face to face with a snake on the path. Her body reacts instantly. (a) Name the type of stress (acute or chronic). (b) Name the division of the nervous system and the part of the adrenal gland that are activated. (c) Name the two hormones that are released.

Q4. A student has been under heavy academic pressure for several months. (a) Name the axis that coordinates the body's response to this ongoing stress. (b) Name the part of the adrenal gland that releases cortisol. (c) State one consequence of prolonged, elevated cortisol.

Q5. Walking to her car at night, Aisha hears footsteps behind her and instantly stops dead, holding her breath and listening intently before deciding what to do. (a) Name the acute stress response shown. (b) Describe what is happening in the body during this response. (c) Explain why this response can be adaptive.

Q6. Distinguish, with an example of each, between: (a) a physiological stress response; (b) a psychological stress response.

Unscaffolded

Q7. Distinguish between an internal stressor and an external stressor. (2 marks)

Q8. A warehouse worker hears a stack of pallets crash to the floor directly behind her and spins around. Explain the biological pathway that produced her acute stress response, from the perception of the threat to the release of the relevant hormones. (4 marks)

- Q9.** Acute stress is described as *self-limiting*, whereas chronic stress is not. Explain what this means for the body in each case, referring to what returns the body to homeostasis after an acute stressor and to what happens while a chronic stressor continues. (4 marks)
- Q10.** Explain the role of cortisol in the chronic stress response, and describe one negative effect of cortisol remaining elevated over a long period. (3 marks)
- Q11.** A student writes: “During a sudden fright, the adrenal cortex releases cortisol, which produces the fight-flight-freeze response.” Identify and correct the error in this statement. (2 marks)
- Q12.** Distinguish between a **stressor** and a **stress response**, using one example to illustrate the difference. (2 marks)
- Q13.** Explain why the freeze response is described as a genuine stress response rather than simply a failure to react, and identify the branch of the autonomic nervous system usually said to dominate during it. (3 marks)
- Q14. Research scenario.** A psychologist investigates whether ongoing work stress is associated with higher cortisol. She recruits 80 working adults: 40 who report **high** ongoing work demands and 40 who report **low** ongoing work demands. Each participant provides a morning saliva sample, and the psychologist measures its cortisol concentration (in nmol/L). (a) Identify the independent variable, the dependent variable and the population. (b) Write a directional research hypothesis for this investigation. (c) The two groups were **not** randomly allocated. Explain why this limits the conclusion the psychologist can draw about whether work stress *causes* higher cortisol. (6 marks)
- Q15. Research scenario.** A student researcher asks 25 volunteers to complete a self-report questionnaire about how stressed they have felt, before and after a six-week relaxation program. (a) Name and explain one ethical concept or guideline the researcher should apply, given the personal nature of the data. (b) The stress scores dropped after the program. Explain one reason why this result may not validly show that the program reduced stress. (4 marks)
- Q16.** Over exam period, Leah studies intensely for three weeks and feels constantly tense (an ongoing pressure). On the morning of her first exam, a fire alarm suddenly goes off and she jumps, her heart pounding. Using the correct pathways, explain how Leah’s body responds to (i) the three weeks of exam pressure and (ii) the sudden alarm. (4 marks)
- Q17. Research scenario.** A study measured the mean morning cortisol concentration of two groups of adults:

Group	Mean cortisol (nmol/L)	Standard deviation (nmol/L)
High chronic stress	18.4	3.1
Low chronic stress	12.7	2.6

- (a) With reference to the data, state which group had the higher mean cortisol, and by how much.
- (b) Name the gland structure and the axis responsible for releasing this hormone.
- (c) Suggest one physiological consequence for the high-stress group if their cortisol remains elevated. (5 marks)
- Q18.** After his family’s home was damaged in a storm, Tom has spent two months dealing with repairs, insurance and disrupted routines. He feels persistently on edge, is often irritable with his family, and has had several colds in a row. Using the concepts of stressors and stress responses, analyse Tom’s situation, identifying the type of stressor, whether the stress is acute or chronic, and one physiological and one psychological response. (4 marks)
- Q19. Extended response.** Nadia has just moved out of home to start university in a new city. In her first week, a dog rushes at her in a park and she leaps out of the way, heart racing. Over the following months she struggles with money, coursework and loneliness, and by mid-semester she is constantly tired, is getting sick often, and feels overwhelmed. Using your knowledge of stressors and the acute and chronic stress responses, explain how Nadia’s body and mind respond both to the **dog in the park** and to the **ongoing pressures** of her first semester. Refer to the relevant pathways, hormones and adrenal structures, and to both physiological and psychological responses. (6 marks)

Answers

Q1. (a) Any event/stimulus (internal or external) that produces a stress response. (b) A stressor that originates within the person (their own thoughts, feelings or physiological state). (c) A stressor that originates outside the person, in the environment. **Q2.** (a) External = losing his job / money worries from his situation; internal = his nightly worrying about money. (b) Psychological = feeling hopeless / avoiding friends. (c) Physiological = trouble sleeping (disrupted sleep) or his tension headaches. **Q3.** (a) Acute. (b) Sympathetic nervous system; adrenal medulla. (c) Adrenaline and noradrenaline. **Q4.** (a) The HPA axis. (b) The adrenal cortex. (c) Suppressed immune function (greater susceptibility to illness). **Q5.** (a) The freeze response. (b) Momentary, tense immobility with heightened alertness — a brief, parasympathetic-dominated pause while the body remains aroused and ready to act. (c) It lets her stop and assess the threat (and avoid drawing attention) before committing to fight or flight. **Q6.** (a) A bodily/biological reaction, e.g. increased heart rate. (b) A cognitive, emotional or behavioural reaction, e.g. feeling anxious. **Q7.** An internal stressor comes from within the person (their own thoughts/body); an external stressor comes from the person's environment. **Q8.** Hypothalamus perceives the threat → activates the sympathetic nervous system → stimulates the adrenal medulla → releases adrenaline and noradrenaline (fast, neural pathway). **Q9.** Acute: arousal is intense but brief — once the threat passes the parasympathetic nervous system restores homeostasis, so the response ends by itself. Chronic: the stressor continues, so the HPA axis keeps cortisol elevated above baseline and the body does not recover; prolonged cortisol suppresses immune function. **Q10.** Cortisol (from the adrenal cortex via the HPA axis) sustains energy (glucose) release to meet ongoing demand; if elevated for a long period it suppresses the immune system, increasing susceptibility to illness. **Q11.** Error: in a sudden fright the adrenal **medulla** releases **adrenaline/noradrenaline** (via the sympathetic nervous system); the adrenal cortex and cortisol are the **chronic** response, not the acute one. **Q12.** A stressor is the trigger — the event or circumstance that places a demand on the person (e.g. an upcoming exam); the stress response is the person's reaction to it (e.g. a racing heart and feeling anxious). **Q13.** Freeze is an active, alert immobility (tense stillness, heightened vigilance), not an absence of response; the parasympathetic nervous system is usually said to dominate. **Q14.** (a) IV = level of ongoing work stress (high vs low); DV = salivary cortisol concentration (nmol/L); population = working adults. (b) "It was hypothesised that working adults with high ongoing work stress would have a higher mean salivary cortisol concentration than working adults with low ongoing work stress." (c) The groups were pre-existing and not randomly allocated, so extraneous variables may differ between them; a relationship can be shown but causation cannot be firmly concluded. **Q15.** (a) Confidentiality (keep responses private and de-identified) — or non-maleficence (avoid harm). (b) There is no control group, so the drop could be due to extraneous variables (e.g. the passage of time, demand characteristics, self-report bias), not the program. **Q16.** (i) Three weeks of pressure = chronic → HPA axis → adrenal cortex → cortisol (sustained). (ii) Sudden alarm = acute → sympathetic nervous system → adrenal medulla → adrenaline/noradrenaline (fast fight-flight-freeze arousal). **Q17.** (a) High chronic stress, by $18.4 - 12.7 = 5.7$ nmol/L. (b) Adrenal cortex; the HPA axis. (c) Suppressed immune function / increased susceptibility to illness (or fatigue, disrupted sleep). **Q18.** External stressor (storm damage and its aftermath); chronic stress; physiological = repeated colds (suppressed immunity from prolonged cortisol); psychological = persistent edginess/irritability. **Q19.** Model response given in the solutions.

Fully-worked solutions

Q1. (a) A **stressor** is any event, object or circumstance — internal or external — that **produces a stress response** (that places a demand on the person). (b) An **internal stressor** originates **within the person**, arising from their own thoughts, feelings, attitudes or physiological state (for example, worry or illness). (c) An **external stressor** originates **outside the person**, in their **environment** (for example, an exam or a demanding job).

Q2. (a) The **external stressor** is the loss of his job and the resulting money problems, which come from Sam's environment; the **internal stressor** is his own nightly **worrying about money**, which arises within him (in his own thoughts). (b) A **psychological** stress response is feeling **hopeless** (an emotional reaction) or **avoiding his friends** (a behavioural reaction). (c) A **physiological** stress response is his **trouble sleeping** or his **tension headaches** — automatic **bodily** changes produced by the ongoing stress.

Q3. (a) This is **acute** stress — the snake is a sudden, immediate threat producing brief, intense arousal. (b) The **sympathetic nervous system** is activated, which stimulates the **adrenal medulla** (the inner part of the adrenal glands). (c) The adrenal medulla releases **adrenaline** and **noradrenaline**.

Q4. (a) Ongoing stress is coordinated by the **HPA axis** (hypothalamic–pituitary–adrenal axis). (b) Cortisol is released by the **adrenal cortex** (the outer part of the adrenal glands). (c) One consequence of prolonged, elevated cortisol is **suppression of the immune system**, which increases the student’s susceptibility to illness (other acceptable effects: fatigue, disrupted sleep, reduced wellbeing).

Q5. (a) Aisha shows the **freeze** response. (b) Her body becomes **momentarily immobile and tense while remaining highly alert** — she stops, holds still and focuses intently on the potential threat. This is usually described as a brief, **parasympathetic-dominated** state of stillness, even though the body remains physiologically aroused and ready to switch to fight or flight. (c) Freezing is **adaptive** because it lets her **pause and gather information** about the threat (and avoid drawing attention to herself) before committing to an action, improving her chances of responding effectively.

Q6. (a) A **physiological stress response** is a **bodily/biological** reaction to a stressor — for example, an **increased heart rate** (or sweating, muscle tension, raised cortisol). (b) A **psychological stress response** is a **cognitive, emotional or behavioural** reaction — for example, feeling **anxious** (or difficulty concentrating, irritability, social withdrawal). The two differ in that one is a bodily change and the other is a mental/behavioural change.

Q7. An **internal stressor** originates **within the person** — from their own thoughts, feelings or physiological state (such as worrying or being unwell). An **external stressor** originates **outside the person**, in their **environment** (such as an exam or a noisy workplace). The two are contrasted by their **source**: inside the person versus in the environment.

Q8. The crash behind her is a **sudden threat**, so the **hypothalamus** in the brain perceives the threat and activates the **sympathetic nervous system** (the arousing branch of the autonomic nervous system). The sympathetic nervous system stimulates the **adrenal medulla** — the **inner** part of the adrenal glands — which releases the hormones **adrenaline** and **noradrenaline** into the bloodstream. Because this pathway is largely **neural**, it acts within **seconds**, producing the rapid arousal (an increased heart rate, blood redirected to the muscles) that lets the worker turn and react almost instantly.

Q9. Acute stress is **self-limiting** because the stressor itself is **sudden and short-lived**: it produces an **intense but brief** burst of arousal, and once the threat has passed the **parasympathetic** nervous system takes over and returns the body toward its **normal resting state (homeostasis)**. The response therefore switches itself off, and the person’s heart rate, breathing and hormone levels come back down to baseline within minutes. Chronic stress is **not** self-limiting because the stressor **continues**: the **HPA axis** keeps stimulating the release of **cortisol**, so arousal stays **above baseline** for weeks or months and the body never fully returns to homeostasis. The consequence is that the very responses that are adaptive in the short term become **harmful** — **prolonged elevated cortisol suppresses the immune system** and contributes to fatigue and reduced wellbeing.

Q10. Cortisol is released from the **adrenal cortex** via the **HPA axis** during **chronic (ongoing)** stress. Its role is to keep the body supplied with **energy** — it increases the availability of **glucose** in the blood — so the person can keep meeting a sustained demand, and it dampens non-essential functions. However, if cortisol remains **elevated over a long period**, it **suppresses the immune system**, leaving the person **more susceptible to illness and infection** (and contributing to fatigue and poorer wellbeing).

Q11. The statement is **incorrect**. During a **sudden fright** (an **acute** stressor), it is the **adrenal medulla** — not the cortex — that is stimulated, and it releases **adrenaline** and **noradrenaline** (via the **sympathetic nervous system**) to produce the fight-flight-freeze response. The **adrenal cortex** and **cortisol** (via the **HPA axis**) are involved in the **chronic** stress response, not the acute one. The statement wrongly swaps the chronic pathway into an acute situation.

Q12. A **stressor** is the **trigger** — any event, object or circumstance (internal or external) that places a demand on the person and **produces** a stress response, for example an **upcoming exam**. A **stress response** is the person’s **reaction** to that stressor — the physiological and psychological changes that follow, for example a **racing heart** and **feeling anxious** before the exam. The two are distinguished as **cause and effect**: the stressor is the demand the person faces, whereas the stress response is what happens **within the person’s body and mind** because of it.

Q13. The **freeze** response is a genuine stress response because it is an **active, alert immobility**, not an absence of reaction: the person becomes **tensely still and hypervigilant**, focusing on the threat and holding position while the body remains aroused and ready to switch to fight or flight. This “keeping still” can be adaptive (for example, to avoid detection or to gather information first). The branch of the autonomic nervous system usually said to **dominate** during freezing is the **parasympathetic** nervous system, which produces the immobility.

Q14. (a) The **independent variable** is the **level of ongoing work stress** (high versus low); the **dependent variable** is the **salivary cortisol concentration** (measured in nmol/L); the **population** is **working adults** (the 80 recruited adults are the sample drawn from this population). (b) A suitable **directional hypothesis** is: *“It was hypothesised that working adults with high ongoing work stress would have a higher mean salivary cortisol concentration than working adults with low ongoing work stress.”* (This names **both levels** of the IV and states a **directional** prediction for the DV.) (c) Because the two groups were **pre-existing and not randomly allocated**, they may differ on **extraneous (participant) variables** other than work stress — for example age, health, sleep or diet — which could also affect cortisol. The study can therefore show an **association** between reported work stress and cortisol, but it **cannot firmly conclude that work stress causes** the higher cortisol, because a confounding variable may be responsible.

Q15. (a) A relevant ethical guideline is **confidentiality**: because the data concern how stressed people feel (personal, sensitive information), the researcher must keep participants’ responses **private and de-identified**, storing them securely and not disclosing individual results. (An acceptable alternative is the ethical concept of **non-maleficence** — minimising the risk of harm or distress, for example by allowing participants to skip questions and offering support.) (b) The result may **not validly** show the program worked because there was **no control group**: the drop in stress scores could be caused by **extraneous variables** rather than the program — for example the natural **passage of time**, **demand characteristics** (participants guessing the aim and reporting lower stress to “help”), or **self-report bias**. Without comparing to a group that did not do the program, the improvement cannot be confidently attributed to it.

Q16. (i) The **three weeks of exam pressure** are an **ongoing** stressor, so Leah’s body responds through the **chronic** pathway: the **HPA axis** (hypothalamus → pituitary → adrenal cortex) releases **cortisol** from the **adrenal cortex**, keeping her energised but leaving her persistently tense and, over time, at risk of immune suppression. (ii) The **sudden fire alarm** is an **acute** stressor, so her body responds through the **acute** pathway: the **sympathetic nervous system** stimulates the **adrenal medulla** to release **adrenaline** and **noradrenaline**, producing the rapid fight-flight-freeze arousal (jumping, pounding heart) within seconds.

Q17. (a) The **high chronic stress** group had the higher mean cortisol — **18.4 nmol/L** compared with **12.7 nmol/L** for the low-stress group, a difference of $18.4 - 12.7 = 5.7$ nmol/L. (b) The hormone (cortisol) is released by the **adrenal cortex** (the outer part of the adrenal glands), under the control of the **HPA axis**. (c) If the high-stress group’s cortisol **remains elevated**, a likely physiological consequence is **suppression of the immune system**, increasing their susceptibility to illness (other acceptable answers: fatigue, disrupted sleep, other long-term health effects).

Q18. The damage to Tom’s home and its two-month aftermath (repairs, insurance, disrupted routines) is an **external stressor** — it comes from his environment. Because it is **prolonged and ongoing**, Tom is experiencing **chronic stress**, which works through the **HPA axis** and sustained **cortisol** release from the **adrenal cortex**. A **physiological** stress response is his run of **repeated colds**, consistent with prolonged cortisol **suppressing his immune system**. A **psychological** stress response is his **persistent edginess and irritability** with his family (an emotional/ behavioural reaction). Together these show a chronic stress response with both bodily and psychological components.

Q19. Model response (6 marks). Nadia faces two very different kinds of stressor, and her body responds through two different pathways.

- **The dog in the park (acute).** The dog rushing at her is a **sudden, external stressor**, producing an **acute** stress response. Her **hypothalamus** perceives the threat and activates the **sympathetic nervous system**, which stimulates the **adrenal medulla** (the inner adrenal gland) to release **adrenaline** and **noradrenaline**. Within seconds this produces intense **physiological** arousal — a **racing heart**, faster breathing and blood redirected to her muscles — enabling the **flight** response of leaping out of the way. Psychologically she feels a sudden jolt of **fear**. Once the danger passes, her **parasympathetic** nervous system restores her to calm.
- **The pressures of first semester (chronic).** Her ongoing **money, coursework and loneliness** problems are **prolonged stressors** (a mix of external demands and internal worry), producing **chronic** stress. These are managed by the **HPA axis**: her **hypothalamus** signals the **pituitary gland**, which prompts the **adrenal cortex** (the outer adrenal gland) to release **cortisol**. Sustained high cortisol keeps energy available but, over months, **suppresses her immune system** — explaining why she is **getting sick often** and is **constantly tired** (physiological responses) — while **psychologically** she feels **overwhelmed** and low.

In short, Nadia’s acute response to the dog runs quickly through the **sympathetic nervous system and adrenal medulla (adrenaline/noradrenaline)**, whereas her chronic response to the semester’s demands runs slowly through the **HPA axis and adrenal cortex (cortisol)**; both involve **physiological** and **psychological** components. (Full marks

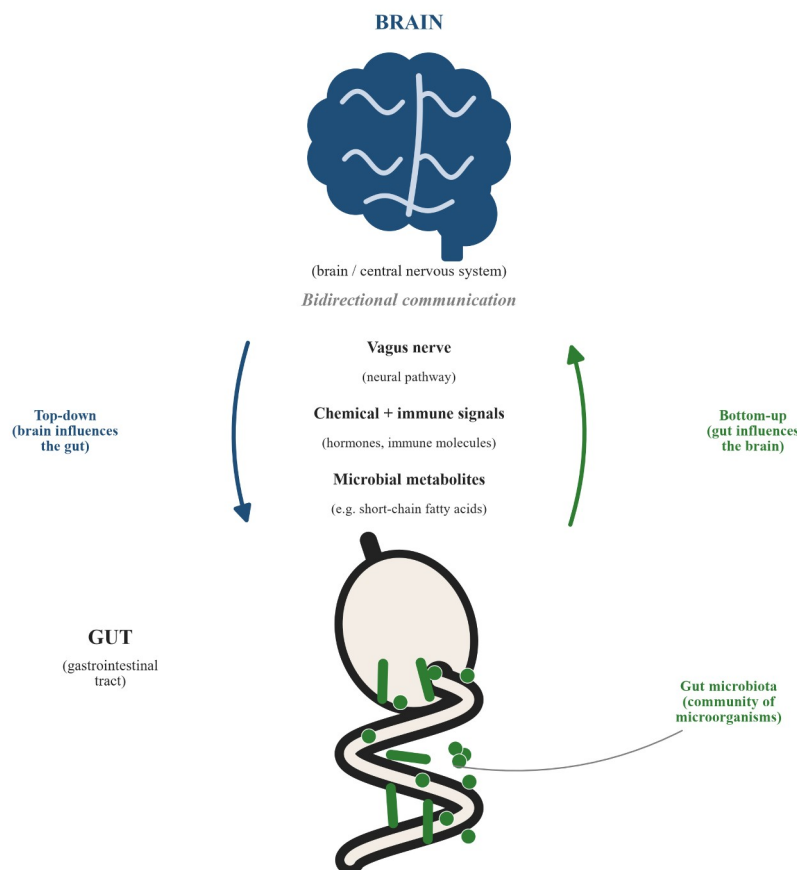
require: correct identification of the acute vs chronic stressors; the correct pathway, hormone and adrenal structure for each; and at least one physiological and one psychological response.)

Theory

Stress is not only a matter of the brain and the nervous system. A growing body of research suggests that the **gut** — the gastrointestinal tract and the community of microorganisms living inside it — is in constant, two-way communication with the brain, and that this communication may help to shape our mood, our behaviour and the way we respond to stress. The system that links the two is called the **gut–brain axis**. It is an **area of emerging research**: the ideas are promising and are being actively investigated, but much of the evidence is still new, and a good deal of it comes from animal studies or is correlational, so conclusions must be drawn cautiously.

The **gut–brain axis (GBA)** is the **bidirectional (two-way) communication network** that links the **gut and its microbiota** with the **brain and central nervous system**. “Bidirectional” means the signals travel in **both directions**: the brain can influence the gut, and the gut can influence the brain.

The **gut microbiota** is the **community of microorganisms** — chiefly bacteria, but also other microbes — that live in the gastrointestinal tract, especially the intestines. An individual’s gut is home to an enormous number and variety of these microorganisms. The particular mix a person carries (how many different types, and in what balance) can differ from person to person and can change over time with factors such as diet, illness, medication and stress.



How the gut and brain communicate. The gut–brain axis is not a single structure but a set of **pathways** that carry signals between the gut and the brain in both directions:

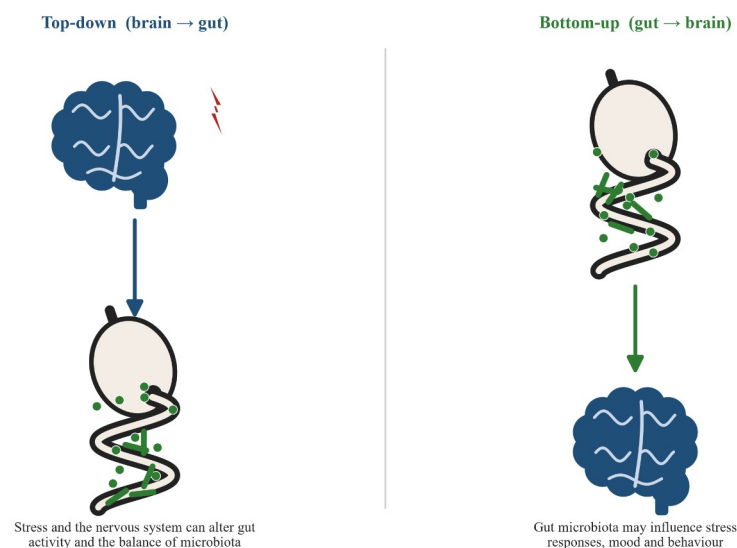
- **A neural pathway — the vagus nerve.** The **vagus nerve** is a major nerve that provides a **direct, two-way neural link** between the gut and the brain, carrying signals up to the brain and down from it. (The gut also has its own extensive network of neurons lining the gastrointestinal tract, which communicates with the central nervous system largely through the vagus nerve.)
- **Chemical and immune signals.** The gut and brain also communicate through the bloodstream using chemical messengers. These include **hormones** (such as the stress hormone cortisol), and **immune signalling**

molecules released as part of the body's inflammatory response. Activity in one part of the system can change the level of these messengers and so affect the other part.

- **Microbial metabolites.** The gut microbiota are chemically active: as they break down food they **produce their own molecules** (for example, short-chain fatty acids), and they can influence the production of **neurotransmitters and their building blocks** (such as serotonin and GABA) in the gut. These microbial metabolites are thought to be one of the ways the microbiota may influence signalling to the brain.

Two directions of influence. Because the communication is bidirectional, it is useful to separate the two directions (see the figure below).

- **Top-down (brain → gut).** The brain and nervous system can alter what happens in the gut. When a person experiences stress, for example, signals from the brain can change **gut activity** (such as the speed of digestion) and, over time, may alter the **balance of the microbiota**.
- **Bottom-up (gut → brain).** The gut and its microbiota can, in turn, send signals that **may influence** the brain — and with it a person's **mood, stress responses and behaviour**. This bottom-up direction is the more surprising and the more heavily researched idea, because it suggests that microorganisms in the gut might play a part in psychological processes.



Because signals run both ways, the gut and brain can form a **feedback loop**: stress (top-down) may shift the microbiota, and the altered microbiota may then feed back (bottom-up) to affect how a person feels and copes — which can itself be a further source of stress.

Why the gut–brain axis is described as “emerging” research. The gut–brain axis is an area of **emerging and evolving evidence**, and this status is itself examinable. Several features explain why:

- **Much of the strongest evidence comes from animal models.** Many key findings come from studies of **mice and rats** — for example, animals raised to have no gut microbiota, or animals whose microbiota have been altered, which then behave differently under stress. Animal studies allow tight experimental control, but their results **may not generalise** directly to humans, whose biology, diet and experience are far more complex.
- **Much of the human evidence is correlational.** Studies in people often **measure** the microbiota and **measure** mood or stress and look for an **association** between them. A correlation shows the two are related, but it **cannot establish cause and effect** on its own, and it cannot show the **direction** of any influence (a difference in the microbiota might cause a change in mood, or a change in mood — and the behaviour that goes with it, such as diet — might change the microbiota, or a third factor might affect both).
- **The mechanisms are not yet fully mapped, and results are mixed.** Exactly *how* microbial signals reach and affect the brain is still being worked out, and studies that try to change the microbiota (for example, with **probiotics** — live microorganisms taken to alter the gut) have produced **inconsistent results**, often with small samples.

For all these reasons, careful writers use **cautious, hedged language** about the gut–brain axis. It is accurate to say the gut microbiota **may influence, is associated with, or appears to be linked to** stress and behaviour; it is **not yet**

accurate to say the microbiota **causes** a particular mood or **cures** a disorder. Being able to describe the gut–brain axis *and* evaluate the strength of the evidence for it is exactly the kind of scientific-reasoning skill this topic is designed to develop.

Key points to keep straight.

- The gut–brain axis is **bidirectional** — brain → gut (top-down) **and** gut → brain (bottom-up).
- The **gut microbiota** is the community of microorganisms in the gut; it is one part of the axis, not the whole axis.
- The main pathways are the **vagus nerve** (neural), **chemical and immune signals** (hormones and immune molecules), and **microbial metabolites**.
- The field is **emerging**: much evidence is **animal-based or correlational**, so keep causal claims **hedged**.

Worked examples

Example 1 — scaffolded

Waiting backstage to audition for a place in a youth orchestra, Imogen feels intensely nervous. As her anxiety rises she notices her stomach “churning” and feels slightly nauseous, and she has no appetite for the food she brought with her. Using the gut–brain axis, identify the **direction** of communication shown here, name a **pathway** that could carry the signal, and explain the link.

Working	Comment
The trigger is a psychological state (anxiety) in the brain , and the <i>effect</i> is a change in the gut (churning, nausea, loss of appetite).	Ask which end the signal starts at and which end it acts on. Brain → gut.
This is top-down communication (brain → gut).	Name the direction using the correct term.
The signal could travel via the vagus nerve (a neural pathway) and via stress hormones such as cortisol carried in the blood.	Identify a plausible pathway; both the neural and chemical routes are two-way.
Anxiety in the brain alters gut activity — here, slowing digestion , which is a non-urgent process the stress response de-prioritises — and this produces the physical symptoms Imogen feels.	Link the pathway back to the specific scenario, don’t just define. Match the direction to the fight-flight-freeze account: arousal slows digestion.

Example 2 — scaffolded

In a study, mice raised so that they had **no gut microbiota** showed **exaggerated** behavioural responses to a mild stressor compared with normal mice. When these mice were later given a normal microbiota, their stress responses became more like those of ordinary mice. Identify the **direction** of communication this study investigates, state what the results **suggest**, and explain **one reason for caution** in applying the finding to people.

Working	Comment
The variable being changed is the gut microbiota ; the outcome measured is the stress-related behaviour produced by the brain.	The influence runs from gut to brain.
This investigates bottom-up communication (gut → brain).	Name the direction.
The results suggest that the gut microbiota may influence how strongly the brain and body respond to stress.	Use hedged language — “suggest”, “may influence”, not “proves”.
The study used mice, not humans (an animal model), so the findings may not generalise to people, whose biology and experience are far more complex.	Give a specific, correct reason for caution — this is the examinable evaluation skill.

Example 3 — unscaffolded

Define the gut–brain axis, and explain why it is described as an *area of emerging research*.

The **gut–brain axis** is the **bidirectional communication network** linking the **gut and its microbiota** with the **brain and central nervous system**: signals travel from the brain to the gut (top-down) and from the gut to the brain (bottom-up), carried by the **vagus nerve**, by **chemical and immune signals**, and by **microbial metabolites**. It is described as an **area of emerging research** because the evidence, although promising, is still **new and developing**: much of the strongest evidence comes from **animal models** (such as mice) that may not generalise to humans, much of the human evidence is **correlational** and so cannot establish cause and effect, and the **mechanisms are not yet fully understood**, with intervention studies producing **mixed results**. ∴ the gut–brain axis is a genuine, actively investigated link between gut and brain, but one whose influence on behaviour is still being established, so claims about it should be expressed cautiously.

Example 4 — unscaffolded

A wellness blog states: “Science has proven that eating probiotic yoghurt cures anxiety by fixing your gut bacteria.” Evaluate this claim with reference to the current state of gut–brain-axis research.

The claim **overstates** what the evidence shows. It is **plausible**, and consistent with emerging research, that the gut microbiota **may influence** mood and stress responses through the gut–brain axis, and some studies have found associations between the microbiota and anxiety. However, the claim is **not justified** for several reasons. First, the research is **emerging** and much of it is **animal-based or correlational**, so it **cannot prove** that changing gut bacteria *causes* a change in anxiety. Second, intervention studies using probiotics have produced **inconsistent results**, so it is wrong to say a single food “**cures**” anxiety. Third, the word “**cures**” implies a reliable, established treatment, which the current evidence does not support. A more accurate statement would be that the gut microbiota **may be linked to** mood and that this is a **developing** area of study. ∴ the blog’s claim is inaccurate because it treats emerging, largely correlational evidence as if it were settled proof of a cause-and-effect cure.

Practice questions

Scaffolded

Q1. Define each of the following: (a) the gut microbiota; (b) the gut–brain axis; (c) what is meant by describing the communication in the gut–brain axis as *bidirectional*.

Q2. During a stressful week of exams, Priya notices that her digestion changes and her stomach feels unsettled. (a) State whether this is an example of top-down or bottom-up communication. (b) Name one pathway that could carry the signal from her brain to her gut. (c) Explain the link between her stress and her gut symptoms.

Q3. A researcher reports that, in laboratory rats, altering the balance of the gut microbiota was followed by changes in the rats’ anxiety-like behaviour. (a) State whether this concerns top-down or bottom-up communication. (b) Identify what is being changed and what is being measured. (c) Explain, using cautious language, what the finding *suggests*.

Q4. For the gut–brain axis: (a) name the neural pathway that provides a two-way link between the gut and the brain; (b) name two chemical or immune signals that can carry information between the gut and the brain; (c) explain what is meant by *microbial metabolites*.

Q5. The gut–brain axis is described as an area of emerging research. (a) State one reason much of the evidence is considered preliminary. (b) Explain why findings from studies on mice must be applied to humans with caution. (c) Rewrite the following overclaim in appropriately cautious language: “Gut bacteria control your mood.”

Q6. When a person experiences ongoing stress, the gut–brain axis can act as a feedback loop. (a) Describe the top-down step in this loop. (b) Describe the bottom-up step in this loop. (c) Explain why this is described as a *loop* rather than a one-way effect.

Unscaffolded

Q7. Distinguish between top-down and bottom-up communication in the gut–brain axis. (2 marks)

Q8. Describe the role of the vagus nerve in the gut–brain axis. (2 marks)

Q9. Explain what the gut microbiota is, and identify one way it may send signals that reach the brain. (2 marks)

Q10. After a chest infection, Dan takes a course of antibiotics that substantially reduces the diversity of his gut microbiota. Over the following weeks he reports feeling unusually low and irritable. Using bottom-up communication in the gut–brain axis, explain how his change in mood *could* be related to the change in his microbiota, and use appropriately cautious language. (3 marks)

Q11. Explain why the gut–brain axis is described as an *area of emerging research*. (3 marks)

Q12. A student writes: “In a survey, adults who ate more high-fibre food had a more varied gut microbiota and reported fewer symptoms of stress. This shows that eating more fibre reduces stress.” Identify and explain the flaw in this reasoning. (2 marks)

Q13. Research scenario. A psychologist investigates whether adding a **fermented milk drink** containing live bacteria to the diet changes how stressed people feel. Forty-eight university staff volunteer and are randomly allocated to two groups. For six weeks, one group drinks a serve of the fermented drink each morning; the other drinks a serve of a **heat-treated** version of the same drink, which looks and tastes the same but contains no live bacteria. Neither the volunteers nor the psychologist who scores the questionnaires knows which drink a volunteer received. At the end of the six weeks, each volunteer completes a stress questionnaire scored out of 40. (a) Identify the independent variable and the dependent variable. (b) Explain the purpose of the heat-treated drink, and of keeping both the volunteers and the scoring psychologist unaware of who received it. (c) Identify the population this sample is intended to represent, and state one reason the results might not generalise to that population. (d) Name one ethical consideration the researcher must address. (6 marks)

Q14. Research scenario. Much of the evidence that the gut microbiota can influence stress responses comes from experiments on mice, because the microbiota can be tightly controlled in animals in ways that would be impossible in humans. (a) State one advantage of using an animal model to study the gut–brain axis. (b) Evaluate whether findings from mice can be assumed to apply to humans. (4 marks)

Q15. Research scenario. In a small study, 20 participants drank either a probiotic drink or a placebo drink each day for four weeks, after which their anxiety was rated on a 0–50 scale. The mean results are shown below.

Group	Mean anxiety score (/50)	Standard deviation
Probiotic drink	18	7
Placebo drink	23	8

- (a) State which group reported the lower mean anxiety, and by how much.
- (b) The study is repeated with 200 participants instead of 20. State whether this mainly reduces *random* or *systematic* error, and explain what it does to the **precision** of each group’s mean.
- (c) Distinguish between a *conclusion* and an *implication* that could be drawn from this study. (4 marks)

Q16. Evaluate the claim that the gut microbiota influences stress responses in humans. In your answer, refer to both a strength and a limitation of the current evidence, and make a judgement. (4 marks)

Q17. For six months Sunita has worked long hours to meet a series of deadlines. She notices that her digestion has changed and that she has begun to feel flat and on edge. Using the gut–brain axis, explain how Sunita’s stress and her gut could be acting as a feedback loop, naming the direction of each step. (3 marks)

Q18. Explain why a correlational study that finds an association between a person’s gut microbiota and their level of stress cannot, on its own, establish that the microbiota *causes* the stress. (3 marks)

Q19. Extended response. “The gut–brain axis is a promising but still-emerging explanation of how the gut may influence stress and behaviour.” Using the bidirectional nature of the gut–brain axis, and with reference to the current state of the evidence, discuss this statement. (6 marks)

Answers

Q1. (a) The community of microorganisms (mainly bacteria) living in the gastrointestinal tract. (b) The bidirectional communication network linking the gut and its microbiota with the brain and central nervous system. (c) Signals travel in both directions — brain to gut and gut to brain. **Q2.** (a) Top-down. (b) The vagus nerve (or stress hormones such as cortisol). (c) Stress in the brain alters gut activity via the axis, producing her gut symptoms. **Q3.** (a) Bottom-up. (b) Changed = the gut microbiota; measured = the rats' anxiety-like behaviour. (c) It *suggests* the microbiota *may influence* stress/anxiety-related behaviour. **Q4.** (a) The vagus nerve. (b) Hormones (e.g. cortisol) and immune-signalling molecules released in the inflammatory response. (c) Molecules produced by the gut microbiota (e.g. short-chain fatty acids) that may affect signalling to the brain. **Q5.** (a) Much evidence is animal-based or correlational / mechanisms not fully understood / mixed results. (b) Mice differ from humans, so results may not generalise. (c) e.g. "Gut bacteria may influence mood" / "are associated with mood". **Q6.** (a) Stress in the brain alters gut activity and the microbiota (brain → gut). (b) The altered microbiota then feeds back to influence mood and stress responses (gut → brain). (c) Because each direction affects the other, so effects can cycle rather than run one way. **Q7.** Top-down = brain influences the gut; bottom-up = the gut (and its microbiota) influences the brain. **Q8.** The vagus nerve is a neural pathway providing a direct, two-way link that carries signals between the gut and the brain. **Q9.** The gut microbiota is the community of microorganisms in the gut; it may signal to the brain via microbial metabolites (or by acting on the vagus nerve or immune/chemical signals). **Q10.** The antibiotics reduced his microbiota diversity; via bottom-up communication this altered microbiota *may* have changed signalling to his brain, which *could* contribute to his low mood — though this cannot be established with certainty. **Q11.** Because much evidence is animal-based or correlational, mechanisms are not fully understood, and intervention results are mixed, so conclusions remain tentative. **Q12.** The reasoning confuses correlation with causation: a survey shows only an association, not that eating more fibre *causes* the lower stress. **Q13.** (a) IV = which drink is taken — the fermented drink with live bacteria vs the heat-treated drink without them; DV = the stress questionnaire score (/40). (b) The heat-treated drink is a placebo, so it controls for the placebo effect; keeping both parties unaware makes the study double-blind, also controlling participant and experimenter expectancy. (c) Population = adults; the 48 are self-selected volunteers from one workplace, so may be unrepresentative (age/occupation/diet). (d) e.g. informed consent (also: right to withdraw, confidentiality). **Q14.** (a) Tight experimental control of the microbiota / cause-and-effect testing not possible in humans. (b) Advantage is control, but mice differ biologically and behaviourally from humans, so findings may not generalise; a judgement that animal evidence is suggestive but not conclusive for humans. **Q15.** (a) Probiotic group, by 5 points (18 vs 23). (b) Random error, because averaging over more participants cancels out chance variation, making each group mean more precise; it does not fix a consistent (systematic) bias. (c) Conclusion = a statement about whether the IV affected the DV in this study; implication = the broader real-world meaning/application of that result. **Q16.** Strength: converging animal and human studies suggest an association. Limitation: much evidence is animal-based/correlational, so causation is not established. Judgement: the influence is plausible but not yet proven in humans. **Q17.** Sunita's prolonged stress (top-down) alters her gut activity and microbiota; the altered microbiota (bottom-up) may then affect her mood and stress responses, which can further raise her stress — a loop. **Q18.** A correlation shows only that the two vary together; it cannot show direction, and a third variable may explain both, so cause is not established. **Q19.** Model response given in the solutions.

Fully-worked solutions

Q1. (a) The **gut microbiota** is the **community of microorganisms** — mainly bacteria, but also other microbes — that live in the **gastrointestinal tract**, especially the intestines. (b) The **gut–brain axis** is the **bidirectional communication network** that links the **gut and its microbiota** with the **brain and central nervous system**. (c) Describing the communication as **bidirectional** means signals travel in **both directions** — from the brain to the gut (top-down) and from the gut to the brain (bottom-up) — not one way only.

Q2. (a) This is **top-down** communication: the effect runs from the **brain** (stress/anxiety) to the **gut**. (b) The signal could be carried by the **vagus nerve** (a neural pathway) or by **stress hormones** such as cortisol in the bloodstream. (c) When Priya is stressed, her **brain** sends signals via the gut–brain axis that **alter her gut activity** (for example, changing the speed of digestion), which produces the unsettled stomach she notices. The stress in the brain is the cause and the gut change is the effect.

Q3. (a) It concerns **bottom-up** communication, because the influence runs from the **gut microbiota** to the **brain/behaviour**. (b) What is being **changed** (manipulated) is the **balance of the gut microbiota**; what is being

measured is the rats' **anxiety-like behaviour**. (c) The finding **suggests** that the gut microbiota **may influence** anxiety-related behaviour — but because this is an animal study, it can only be stated cautiously, not as proof for humans.

Q4. (a) The **vagus nerve** provides the two-way neural link between the gut and the brain. (b) The **two** chemical or immune signals are **hormones** (such as the stress hormone cortisol) and **immune-signalling molecules** produced in the inflammatory response. (Microbial metabolites are counted separately, as the third pathway, and are the subject of part (c).) (c) **Microbial metabolites** are **molecules produced by the gut microbiota** (for example, short-chain fatty acids) as they break down food; these chemicals are one proposed way the microbiota may **influence signalling to the brain**.

Q5. (a) Much of the evidence is **animal-based or correlational**, the **mechanisms are not yet fully understood**, and intervention studies have given **mixed results** (any one). (b) **Mice differ from humans** in their biology, diet and experience, so a result found in mice **may not generalise** to people; animal findings are suggestive rather than conclusive for humans. (c) An appropriately cautious version is: “**Gut bacteria may influence mood**” or “**Gut bacteria are associated with mood**” — using “may” or “associated with” rather than “control”.

Q6. (a) The **top-down** step: ongoing **stress in the brain** sends signals through the axis that **alter gut activity and the balance of the microbiota**. (b) The **bottom-up** step: the **altered microbiota** then sends signals that **may influence** the person's **mood and stress responses**. (c) It is a **loop** because each direction affects the other — the brain changes the gut, and the changed gut feeds back to the brain — so the effects can **cycle** and reinforce one another rather than running in a single direction once.

Q7. In **top-down** communication the **brain and nervous system influence the gut** — for example, stress altering gut activity and the microbiota. In **bottom-up** communication the **gut and its microbiota influence the brain** — for example, the microbiota affecting mood or stress responses. The two differ in the **direction** the signal travels. (Both directions named for the two marks.)

Q8. The **vagus nerve** is a major nerve that provides a **direct, two-way (bidirectional) neural pathway** between the gut and the brain. It **carries signals from the brain down to the gut and from the gut up to the brain**, making it one of the main routes of communication in the gut–brain axis.

Q9. The **gut microbiota** is the **community of microorganisms (mainly bacteria) living in the gastrointestinal tract**. One way it may send signals that reach the brain is by producing **microbial metabolites** (such as short-chain fatty acids) — or by influencing the **vagus nerve** or **immune and chemical signals** — that may in turn affect the brain.

Q10. The antibiotics **reduced the diversity of Dan's gut microbiota**. Through **bottom-up** communication in the gut–brain axis, this altered microbiota **may** have changed the signals sent to his brain — for example via **microbial metabolites** or the **vagus nerve** — and this **could** have contributed to his low, irritable mood. It is important to use **cautious language** here: because the evidence for the gut–brain axis is emerging and largely correlational, and because his mood could also be affected by the infection itself or by other factors, we can say the microbiota change **may be linked to** his mood, but we **cannot conclude** that it definitely caused it.

Q11. The gut–brain axis is described as **emerging research** because the evidence, though promising, is still **developing** and **not yet conclusive**. First, much of the strongest evidence comes from **animal models** (such as mice), which **may not generalise** to humans. Second, much of the human evidence is **correlational**, so it **cannot establish cause and effect** or the direction of any influence. Third, the **mechanisms are not fully understood** and intervention studies (for example with probiotics) have produced **mixed results**. Because of this, findings are stated tentatively rather than as established fact.

Q12. The flaw is that the student has **confused correlation with causation**. The survey only **measured** the variables — it did not manipulate anyone's diet — so it shows that a higher-fibre diet is **associated with** a more varied microbiota and fewer stress symptoms, but it **does not show that eating more fibre makes stress fall**. The relationship could run the other way (people who are less stressed may find it easier to shop for and cook higher-fibre meals), or a **third variable** — overall diet quality or physical activity, for instance — could influence both. On this evidence the only justified statement is that the two are **related**, not that one produces the other.

Q13. (a) The **independent variable** is **which drink the volunteer receives — the fermented drink containing live bacteria, or the heat-treated drink without them**. The **dependent variable** is the volunteer's **stress questionnaire score (out of 40)**. (b) The **heat-treated drink** is a **placebo**: it looks and tastes the same but lacks the active ingredient, so it controls for the **placebo effect** (improvement produced merely by *believing* one is being treated). Keeping **both the volunteers and the scoring psychologist** unaware of who received which drink makes the study **double-blind**, which additionally controls **experimenter expectancy** (the scorer unconsciously marking the two groups differently). Together these mean any difference between the groups is more likely to be due to the **live bacteria** themselves. (c) The **population** is **adults** (the wider group the researcher wants to draw conclusions about). The results **might not generalise** because the 48 participants are **self-selected volunteers from a single workplace**, so the sample may be unrepresentative in age, occupation, diet or health — and people who volunteer for a diet study may differ from those who do not. (d) One **ethical consideration** is **informed consent** — participants must be told what the study involves, including that they may be given the drink without live bacteria, and agree to take part before it begins. (Other acceptable answers: the **right to withdraw**, **confidentiality**, or ensuring **no harm**.)

Q14. (a) An **advantage** of an **animal model** is that the researcher can **tightly control the gut microbiota** — for example by raising animals with no microbiota, or by adding a specific microbiota — and can run **controlled experiments** that establish cause and effect in ways that would be **impossible or unethical in humans**. (b) However, findings from mice **cannot simply be assumed to apply to humans**. Mice **differ from people** in their biology, their microbiota, their diet and their far simpler behaviour and experience, so a result seen in mice **may not generalise**. The animal evidence is therefore best treated as **suggestive** — it shows the gut–brain link is **plausible and worth investigating in humans** — but it is **not conclusive** proof of the same effect in people. (A judgement that animal findings are informative but must be confirmed in humans earns the evaluation mark.)

Q15. (a) The **probiotic** group reported the **lower** mean anxiety, by **5 points** (18 compared with 23). (b) Repeating the study with more participants mainly reduces **random error**. Random error is unsystematic, chance variation between participants and occasions, scattering scores **either side of the true value**; averaging over **more participants** lets those highs and lows cancel out, so each group mean becomes a more **precise** estimate — repeated samples would cluster more tightly together. It does **not** fix **systematic error**, which is a **consistent bias** built into the method (for example, if the questionnaire's instructions ask people to rate a *typical* day rather than the past week, every score is shifted the same way). Systematic error affects **accuracy** rather than precision, and it stays the same no matter how many participants are added. (c) A **conclusion** is a statement about **whether the independent variable affected the dependent variable in this study** — here, whether the probiotic drink lowered anxiety scores in this sample. An **implication** is the **broader, real-world meaning or application** of that result — for example, what the finding might suggest for how people could support their wellbeing, or what further research it points to. The conclusion stays close to the data; the implication looks beyond it.

Q16. There is a genuine **strength** to the claim: **converging evidence** from both **animal experiments** (where altering the microbiota changes stress-related behaviour) and **human correlational studies** (where the microbiota is associated with stress and mood) points to a **real link**, and there is a **plausible mechanism** (the vagus nerve, chemical and immune signals, and microbial metabolites). However, there is a serious **limitation**: much of the evidence is **animal-based or correlational**, so it **cannot establish that the microbiota causes changes in stress in humans**, and intervention studies have been **mixed** and often small. **Judgement**: on the current evidence it is reasonable to say the gut microbiota **may influence** human stress responses and that this is a **promising, actively investigated** idea, but the causal claim is **not yet proven** and should be stated cautiously.

Q17. Six months of long hours and deadlines is a **chronic stressor**, so the gut–brain axis can act as a **feedback loop** for Sunita. In the **top-down** direction (brain → gut), her prolonged **stress** sends signals through the axis — by way of the **vagus nerve** and **stress hormones** such as cortisol — that **alter her gut activity** (the change she notices in her digestion) and, over time, may **shift the balance of her microbiota**. In the **bottom-up** direction (gut → brain), that **altered microbiota may send signals back that influence her mood and stress responses**, plausibly contributing to feeling flat and on edge. Because feeling flat makes the deadlines harder to meet, that low mood **adds to her stress** and feeds the top-down step again: each direction affects the other, so the effect can **cycle** and reinforce itself rather than running one way once.

Q18. A **correlational study** shows only that two things — the microbiota and the level of stress — **vary together**; it does **not** manipulate one to see its effect on the other. Because of this it **cannot show the direction** of the relationship: a difference in the microbiota might affect stress, or a person's stress (and the diet, sleep and behaviour

that go with it) might change the microbiota. It also cannot rule out a **third variable** (such as diet, illness or lifestyle) that influences **both** at once. Since the study neither controls variables nor establishes direction, it **cannot establish that the microbiota causes the stress** — only that the two are associated.

Q19. Model response (6 marks). The statement is a fair summary of the gut–brain axis: it is a **promising** idea, but the evidence is **still emerging**.

The gut–brain axis is the **bidirectional communication network** linking the **gut and its microbiota** with the **brain and central nervous system**. Its two directions make the idea powerful. In the **top-down** direction, the brain and nervous system can **influence the gut** — for example, stress altering gut activity and, over time, the balance of the microbiota. In the **bottom-up** direction — the more striking claim — the **gut microbiota may influence the brain**, and with it a person's **mood, stress responses and behaviour**, through pathways such as the **vagus nerve, chemical and immune signals** and **microbial metabolites**. Because the communication runs both ways, the gut and brain can form a **feedback loop** in which stress and the microbiota affect each other.

At the same time, the statement rightly calls the explanation **still-emerging**. Much of the strongest evidence comes from **animal models** such as mice, which **may not generalise** to humans; much of the human evidence is **correlational**, and so **cannot establish cause and effect** or its direction; and the **mechanisms are not fully understood**, with probiotic intervention studies giving **mixed results**. For these reasons the influence of the gut on stress and behaviour should be described with **cautious, hedged language** — the microbiota **may influence** or **is associated with** stress, not that it **causes** or **cures** it.

Overall, the gut–brain axis is a **plausible and actively researched** explanation of how the gut may help shape stress and behaviour through bidirectional communication, but because the evidence is still **emerging and largely animal-based or correlational**, its influence on human behaviour is **not yet firmly established**. *(Full marks require: the bidirectional nature explained with both directions; at least one communication pathway named; an accurate account of why the evidence is emerging — animal/ correlational/mixed; appropriately cautious causal language; and a clear overall judgement.)*

Chapter 4 Stress as a psychobiological process — 4C Selye's General Adaptation Syndrome

Theory

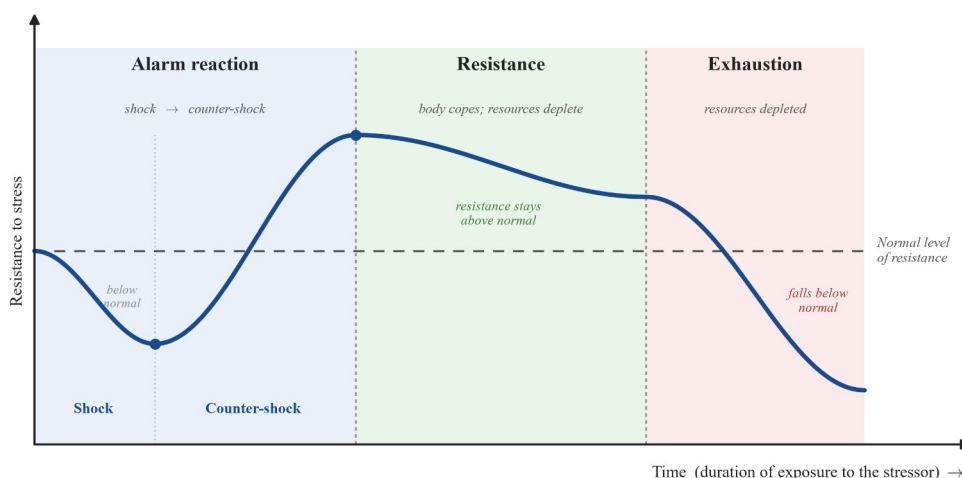
Stress can be examined as a **biological** process — a set of measurable physiological changes the body goes through when it is placed under demand. The most influential **biological model** of stress is **Hans Selye's General Adaptation Syndrome (GAS)**. Working in the mid-twentieth century, Selye exposed laboratory animals (mostly **rats**) to a wide range of stressors — cold, heat, injury, toxins, physical restraint — and noticed that, whatever the stressor, the animals showed the **same underlying pattern** of bodily changes over time. From this he proposed that the body responds to any prolonged stressor through a predictable, three-stage physiological sequence.

The name explains the idea:

- **General** — the response is *non-specific*: the same general pattern of physiological changes occurs regardless of the *type* of stressor.
- **Adaptation** — the body is attempting to **adapt to**, and cope with, the stressor.
- **Syndrome** — the response is a **set of physiological reactions** that occur together in a recognisable sequence.

Because GAS describes stress purely in terms of the body's physiology — hormones, arousal, resources — and makes **no reference to how a person thinks about or interprets the stressor**, it is called a **biological model** of stress.

The three stages of GAS. Selye described three stages: the **alarm reaction** (which has two sub-phases, **shock** and **counter-shock**), **resistance**, and **exhaustion**. Throughout the model, the key measure is the body's **resistance to stress** — its capacity to cope with, and withstand, the demands of the stressor — tracked against a **normal baseline level**.



Stage 1 — Alarm reaction. This is the body's immediate reaction when a stressor is first encountered. It unfolds in two sub-phases:

- **Shock.** For a brief period the body reacts **as though it has been injured** and is momentarily overwhelmed. The body's **resistance to the stressor drops below its normal level**: body temperature and blood pressure fall, and muscle tone decreases. The body has **not yet mobilised** its sympathetic response to the stressor, so its ability to cope is temporarily **below normal**.
- **Counter-shock.** The **sympathetic nervous system** now takes full effect and the body mobilises for action — the **fight–flight–freeze** response. The **adrenal glands release adrenaline** into the bloodstream; heart rate, breathing and blood pressure rise, and energy is directed to the muscles. As a result, the body's **resistance to the stressor climbs above its normal level**, better preparing it to deal with the threat.

Stage 2 — Resistance. If the stressor continues, the body settles into a sustained state of high arousal. **Resistance to the stressor remains above normal.** The adrenal glands release the stress hormone **cortisol**, which keeps blood-glucose (energy) levels elevated and sustains the body's ability to cope. Outwardly the person appears to be **managing well** — they are coping with the ongoing demand. However, maintaining this above-normal resistance is costly: the body's **resources are gradually being depleted**, and sustained high cortisol begins to **suppress the immune system**, leaving the body less able to fight off illness.

Stage 3 — Exhaustion. If the stressor persists for long enough, the body can **no longer maintain** its elevated resistance because its **resources are depleted**. **Resistance to the stressor falls below normal.** The wear and tear of prolonged arousal and elevated cortisol leaves the person fatigued and **vulnerable to illness and stress-related disorders** — for example, high blood pressure, ulcers, weakened immunity, anxiety and low mood. This stage explains why **chronic (prolonged) stress can damage health**.

The explanatory power of GAS. “Explanatory power” means **how well the model actually accounts for stress**. A balanced evaluation weighs its **strengths** against its **limitations**.

Strengths.

- It **identifies a predictable, consistent biological pattern** — a clear three-stage sequence that gives a useful framework for understanding how the body responds to a prolonged stressor.
- It is **grounded in objective physiological evidence**: the bodily changes it describes (arousal, hormone release, resource depletion) are **measurable** and were drawn from **controlled experiments**, giving the model an empirical basis.
- It **explains the link between chronic stress and illness**: the exhaustion stage accounts for *why* prolonged stress leaves people run-down and prone to stress-related disorders, which has real clinical value.

Limitations.

- It was **based largely on animal (rat) studies**, so the findings **may not generalise fully to humans**, whose stress responses are shaped by complex thinking that rats do not share.
- It **ignores psychological and cognitive factors** — in particular a person's **subjective appraisal** of the stressor. Two people facing the same event can respond very differently depending on how **threatening** they judge it to be, but GAS treats the response as purely biological and does not explain these **individual differences**.
- It **assumes the response is non-specific** (a uniform, same reaction to every stressor), yet later research suggests the biological response can **vary with the type of stressor** and with the individual, so the “one general response” claim is an oversimplification.

Because GAS is a *biological* model that leaves out the **psychological** side of stress, it is often contrasted with a psychological account that puts a person's **interpretation** of the stressor at the centre — the approach taken by the **Transactional Model of stress and coping** (studied in 4D). For now, the point to hold onto is that GAS is a **strong biological description** of stress that is **incomplete on its own** because it says nothing about how we *think about* what stresses us.

Key points to keep straight.

- **Shock = resistance below normal** (the body reacts as if injured — faintness, falling temperature and blood pressure, loss of muscle tone); **counter-shock = resistance above normal** (sympathetic arousal). Do not swap these around.
- **Adrenaline release and the fight–flight–freeze response belong to counter-shock, not shock.** Feeling “frozen” is part of *fight–flight–freeze*, so it is a **counter-shock** sign; shock is signalled by the body going faint and limp *before* it mobilises.
- **Resistance stage = above normal, sustained by cortisol, but resources depleting; exhaustion = below normal, resources depleted, vulnerable to illness.**
- GAS is a **non-specific, biological** model — it does **not** account for the person's **cognitive appraisal** of the stressor, which is its central limitation.

Worked examples

Example 1 — scaffolded

Marcus is caught in a house fire. At the first sight of flames he feels faint and light-headed and his legs go weak; seconds later his heart pounds and he sprints outside. Over the following months he is consumed by insurance claims and rebuilding and copes, seeming to hold together well. After nearly a year of unrelenting pressure he becomes constantly exhausted and is repeatedly ill. Map Marcus's experience onto the three stages of Selye's General Adaptation Syndrome.

Working	Comment
The first sight of flames, where Marcus feels faint and his legs go weak , is the shock sub-phase of the alarm reaction — his body reacts as if injured and his resistance momentarily drops below normal .	Alarm reaction begins the moment the stressor is encountered; shock = below normal, body as if injured.
Seconds later his heart pounds and he sprints outside — the counter-shock sub-phase: sympathetic arousal (fight–flight–freeze) lifts his resistance above normal .	Counter-shock = sympathetic activation, resistance rises above normal.
Over the following months he copes with the demands of rebuilding and seems to hold together well — the resistance stage: resistance stays above normal, sustained by cortisol, while resources deplete.	Resistance stage = coping outwardly, but resources are being used up.
After nearly a year he is constantly exhausted and repeatedly ill — the exhaustion stage: resources are depleted, resistance falls below normal and he is vulnerable to illness.	Exhaustion = resources spent, below normal, vulnerability to illness.

Example 2 — scaffolded

A student is unexpectedly asked to give a speech to the whole school. Describe what happens to the student's **resistance to stress** during the **shock** and **counter-shock** sub-phases of the alarm reaction, and explain the role of the sympathetic nervous system.

Working	Comment
In shock , the student is momentarily overwhelmed — they feel faint and light-headed, and their resistance to the stressor drops below its normal level , as the body briefly reacts as if injured.	Name the sub-phase and state the direction of the change (below normal).
The sympathetic response has not yet mobilised — adrenaline has not yet surged — so their capacity to cope is temporarily reduced .	Shock = the body has not yet mobilised its full response.
In counter-shock , the sympathetic nervous system activates the fight–flight–freeze response: adrenaline surges, heart rate and breathing increase and energy is directed to the muscles.	Counter-shock is driven by sympathetic activation.
This raises the student's resistance to the stressor above its normal level , better preparing them to deliver the speech.	Counter-shock = resistance rises above normal.

Example 3 — unscaffolded

Explain why a person can appear to be “coping well” during the **resistance** stage of GAS, yet still be at greater risk of becoming ill.

During the **resistance** stage the body maintains a state of high arousal, keeping its resistance to the stressor **above the normal level**. This is sustained by the release of **cortisol**, which keeps energy (blood glucose) available so the person can continue to meet the demands of the stressor — which is why, from the outside, they appear to be **coping well**.

However, holding resistance above normal is biologically expensive: the body's **resources are steadily depleted**, and prolonged high cortisol **suppresses the immune system**. So even while the person seems to be managing, their capacity to fight off illness is being **eroded**. ∴ apparent coping in the resistance stage masks a gradual depletion of resources and weakening of immunity that raises the risk of illness.

Example 4 — unscaffolded

A school wellbeing poster claims: “Your alarm reaction protects you — from the moment a stressor appears, your body's resistance to it is raised.” Evaluate this claim using Selye's GAS.

The claim is **partly right, but mistimed and overstated**. It is true that the alarm reaction *ends* with the body better prepared: in the **counter-shock** sub-phase the **sympathetic nervous system** mobilises the body for **fight–flight–freeze**, the adrenal glands release **adrenaline**, and resistance to the stressor climbs **above its normal level**. But resistance is **not** raised “from the moment a stressor appears”. The alarm reaction opens with the **shock** sub-phase, in which the body reacts **as though it has been injured** — body temperature and blood pressure fall and muscle tone decreases — so resistance first drops **below normal**, before any mobilisation occurs. The claim also overstates how long the protection lasts: if the stressor persists, GAS predicts the body moves into the **resistance** stage (above normal, sustained by cortisol, resources depleting) and eventually into **exhaustion**, where resources are spent, resistance falls **below normal** and the person becomes vulnerable to illness. ∴ the alarm reaction does lift resistance above normal, but only *after* an initial below-normal dip, and only briefly — so the poster oversimplifies what the model actually predicts.

Practice questions

Scaffolded

Q1. In relation to Selye's General Adaptation Syndrome: (a) state what “GAS” is a model of, and who developed it; (b) name the three stages, in order; (c) name the two sub-phases of the alarm reaction.

Q2. For each event, state which **stage** (or sub-phase) of GAS it best illustrates: (a) after weeks of continuous overtime, a worker is worn out and keeps catching colds; (b) at the instant a car swerves toward them, a cyclist momentarily feels faint and their muscles go limp; (c) moments later, the cyclist's heart pounds and they swerve sharply out of the way.

Q3. For the **alarm reaction**: (a) state what happens to resistance to the stressor during the **shock** sub-phase, relative to normal; (b) state what happens to resistance during the **counter-shock** sub-phase, relative to normal; (c) name the division of the nervous system responsible for the arousal in counter-shock.

Q4. For the **resistance** stage: (a) state whether resistance to the stressor is above or below normal; (b) name the hormone that sustains the body's resistance in this stage; (c) describe what is happening to the body's resources during this stage.

Q5. For the **exhaustion** stage: (a) state whether resistance to the stressor is above or below normal; (b) explain why resistance reaches this level; (c) state one consequence for the person of reaching the exhaustion stage.

Q6. In relation to the explanatory power of GAS: (a) state one **strength** of the model; (b) state one **limitation** of the model; (c) explain why GAS is described as a **biological** model of stress.

Unscaffolded

Q7. Distinguish between the **shock** and **counter-shock** sub-phases of the alarm reaction. (2 marks)

Q8. Describe the **resistance** stage of GAS, with reference to cortisol and the body's resources. (3 marks)

Q9. Explain why the body's resistance to a stressor drops **below normal** during the shock sub-phase of the alarm reaction. (2 marks)

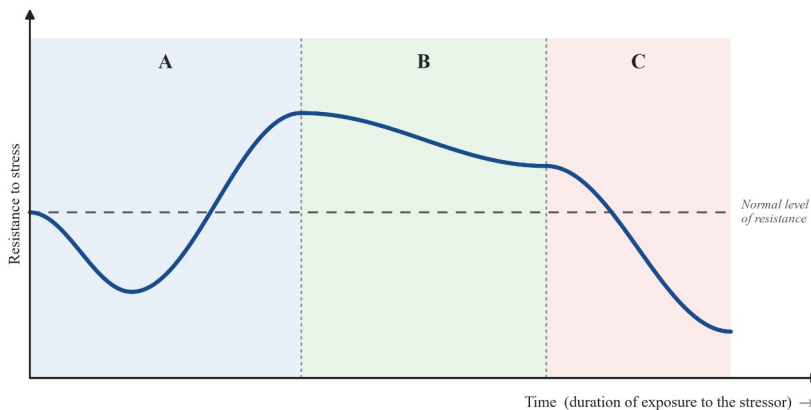
Q10. Leila takes on the full-time care of an unwell parent. On the first night she feels numb and light-headed, then quickly becomes highly alert and manages the emergency. For several months she copes with the daily demands of

caring. Eventually she is chronically fatigued, run-down and frequently unwell. Using the three stages of GAS, explain Leila's physiological experience over this period. (4 marks)

Q11. Explain why the **exhaustion** stage of GAS is associated with an increased vulnerability to illness. Refer to the body's resources and to cortisol in your answer. (3 marks)

Q12. A student writes: "During the shock sub-phase of the alarm reaction, the body's resistance to the stressor rises above its normal level." Identify and correct the error in this statement. (2 marks)

Q13. Stimulus — graph interpretation. The graph below shows a person's resistance to a stressor over time, tracked against their normal level of resistance, as the stressor persists. The three shaded regions A, B and C correspond, in order, to the three stages of GAS.



- (a) Name the stage of GAS shown in region C, and state where resistance ends up in that region relative to the person's normal level.
- (b) Within region A, the curve first dips below the normal line and then rises above it. Name the sub-phase responsible for each part of this movement.
- (c) Explain what the section of the curve in region B represents about the body's functioning. (4 marks)

Q14. Stimulus — research methods. Selye developed GAS after exposing large numbers of **rats** to stressors such as extreme cold and injury, and observing a consistent three-stage physiological pattern. (a) Identify **one strength** of using this kind of controlled animal experiment as the basis for the model. (b) Identify **one limitation** of relying on animal studies when applying GAS to **humans**. (c) Explain how this limitation is connected to GAS's neglect of psychological factors. (4 marks)

Q15. Distinguish between the way GAS explains stress and a **psychological** account that emphasises a person's appraisal of the stressor. Use this to explain why GAS struggles to account for **individual differences** in the stress response. (3 marks)

Q16. Selye claimed that the stress response is "**non-specific**". (a) Explain what "non-specific" means in the context of GAS. (b) Explain why this assumption is considered a limitation of the model. (3 marks)

Q17. Stimulus — research methods. A researcher exposes two groups of laboratory rats to a continuous mild stressor. Group 1 is exposed for 3 days; Group 2 is exposed for 30 days. At the end of the exposure period the researcher measures each rat's blood **cortisol** concentration and examines its immune tissue for signs of damage. (a) Identify the independent variable and the dependent variable(s) in this investigation. (b) Based on GAS, predict which group is more likely to show **depleted resources and immune damage**, and justify your prediction with reference to the relevant stage. (c) Identify one reason why the results of this study should be applied to humans with caution. (4 marks)

Q18. Two colleagues are given the same tight deadline. One treats it as an exciting challenge and feels energised; the other is overwhelmed and dreads it. Explain why Selye's GAS, on its own, cannot fully account for the difference between the two colleagues' experiences. (3 marks)

Q19. *Extended response.* Evaluate the explanatory power of Selye's General Adaptation Syndrome as a model of stress. In your answer, refer to the stages of the model, and discuss both its strengths and its limitations, before reaching a judgement. (6 marks)

Answers

Q1. (a) A biological model of the body's physiological response to stress; developed by Hans Selye. (b) Alarm reaction → resistance → exhaustion. (c) Shock and counter-shock. **Q2.** (a) Exhaustion. (b) Shock (alarm reaction). (c) Counter-shock (alarm reaction). **Q3.** (a) It drops below normal. (b) It rises above normal. (c) The sympathetic nervous system. **Q4.** (a) Above normal. (b) Cortisol. (c) They are gradually being depleted while resistance is sustained. **Q5.** (a) Below normal. (b) The body's resources are depleted, so it can no longer sustain elevated resistance. (c) Increased vulnerability to illness / stress-related disorders (e.g. weakened immunity). **Q6.** (a) e.g. it identifies a predictable biological pattern grounded in physiological evidence. (b) e.g. it ignores psychological/cognitive factors (subjective appraisal). (c) It explains stress purely through physiological changes, without reference to how the person interprets the stressor. **Q7.** In shock, resistance drops below normal (the body reacts as if injured); in counter-shock, sympathetic arousal lifts resistance above normal. **Q8.** Resistance stays above normal; cortisol sustains it by keeping energy available; the person copes outwardly but resources deplete and immunity is suppressed. **Q9.** The body is momentarily overwhelmed and reacts as if injured before it has mobilised its full (sympathetic) response, so its ability to cope is temporarily below normal. **Q10.** Numb/light-headed = shock; highly alert/managing the emergency = counter-shock; months of coping = resistance; chronic fatigue and illness = exhaustion. **Q11.** Prolonged stress depletes the body's resources and sustained high cortisol suppresses the immune system, so resistance falls below normal and the person is more prone to illness. **Q12.** Error: in shock, resistance drops *below* normal, not above; it is in counter-shock that resistance rises above normal. **Q13.** (a) Exhaustion; resistance falls, ending below normal. (b) Dip below = shock; rise above = counter-shock. (c) Region B is the resistance stage: the body is coping with the stressor at an elevated (above-normal) level of resistance, sustained by cortisol, while its resources deplete. **Q14.** (a) e.g. controlled conditions allow the stressor to be manipulated and a consistent pattern to be measured objectively. (b) Rats differ physiologically and cognitively from humans, so findings may not generalise. (c) Because animal studies capture only the biology, they miss the human psychological (appraisal) dimension that GAS also leaves out. **Q15.** GAS explains stress as a fixed biological sequence; a psychological account explains it through the person's appraisal/interpretation of the stressor. Because GAS assumes the same response for everyone, it cannot explain why individuals differ. **Q16.** (a) The same general physiological pattern occurs regardless of the type of stressor. (b) It is a limitation because responses can actually vary with the stressor and the individual, so the assumption oversimplifies. **Q17.** (a) IV = duration of exposure to the stressor (3 vs 30 days); DV = blood cortisol concentration and degree of immune-tissue damage. (b) Group 2 (30 days) — prolonged exposure moves the body toward the exhaustion stage, depleting resources and damaging immunity. (c) Rats are not humans, so the findings may not generalise (and humans' appraisal of stressors also differs). **Q18.** GAS is a non-specific biological model that ignores cognitive appraisal, so it cannot explain why one colleague appraises the deadline as a challenge and the other as a threat. **Q19.** Model response given in the solutions.

Fully-worked solutions

Q1. (a) The **General Adaptation Syndrome (GAS)** is a **biological model** of the body's **physiological response to stress**; it was developed by **Hans Selye**. (b) The three stages, in order, are the **alarm reaction**, then **resistance**, then **exhaustion**. (c) The two sub-phases of the alarm reaction are **shock** and **counter-shock**.

Q2. (a) **Exhaustion** — after prolonged stress the worker's resources are depleted, resistance is below normal and they are prone to illness. (b) **Shock** (the first sub-phase of the **alarm reaction**) — the instant reaction in which the body responds as though injured, so the person feels faint and weak and resistance briefly drops below normal. (c) **Counter-shock** (the second sub-phase of the **alarm reaction**) — sympathetic arousal (the pounding heart, the swerve) lifts resistance above normal.

Q3. (a) During **shock**, the body reacts as though it has been injured and resistance to the stressor **drops below its normal level**. (b) During **counter-shock**, sympathetic arousal mobilises the body, so resistance to the stressor **rises above its normal level**. (c) The **sympathetic nervous system** (of the autonomic nervous system) produces the fight–flight–freeze arousal in counter-shock.

Q4. (a) In the resistance stage, resistance to the stressor is **above normal**. (b) The hormone **cortisol** sustains it, by keeping blood-glucose (energy) elevated. (c) The body's **resources are gradually being depleted** — the above-normal resistance is maintained at a cost, and prolonged cortisol also begins to suppress the immune system.

Q5. (a) In exhaustion, resistance to the stressor is **below normal**. (b) It reaches this level because the body's **resources have been depleted** by prolonged arousal, so it can **no longer maintain** the elevated resistance of the resistance stage. (c) The person becomes **vulnerable to illness and stress-related disorders** — for example, weakened immunity, high blood pressure, ulcers, fatigue, anxiety or low mood (any one).

Q6. (a) A **strength** is that GAS **identifies a predictable biological pattern** of responses that is **grounded in measurable physiological evidence** (and it explains the chronic-stress–illness link). (b) A **limitation** is that it **ignores psychological/cognitive factors** — especially the person's **subjective appraisal** of the stressor (or: it is based on animal studies; it assumes a non-specific response). (c) GAS is a **biological** model because it explains stress **purely through physiological changes** (arousal, hormones, resource depletion) and makes **no reference to how the person thinks about or interprets** the stressor.

Q7. In the **shock** sub-phase the body reacts **as if it has been injured** and its resistance to the stressor **drops below normal**. In the **counter-shock** sub-phase the **sympathetic nervous system** activates the fight–flight–freeze response, so resistance to the stressor **rises above normal**. The two sub-phases therefore move resistance in **opposite directions** — below normal, then above it. (Both sides contrasted for the two marks.)

Q8. In the **resistance** stage the body maintains a state of high arousal and its resistance to the stressor stays **above normal**. This is sustained by the release of **cortisol**, which keeps energy (blood glucose) available so the person can continue to cope with the ongoing stressor. Although the person appears to be managing, this comes at a cost: the body's **resources are gradually depleted**, and sustained cortisol begins to **suppress the immune system**.

Q9. In the **shock** sub-phase the body is momentarily **overwhelmed** and reacts **as though it has been injured** — body temperature and blood pressure fall. This happens **before** the body has mobilised its full **sympathetic** response, so for a brief period its ability to cope with the stressor is **reduced**, and its resistance falls **below the normal level**.

Q10. *Shock*: on the first night, feeling **numb and light-headed** is the **shock** sub-phase of the alarm reaction — Leila's resistance briefly drops **below normal**. *Counter-shock*: becoming **highly alert and managing the emergency** is the **counter-shock** sub-phase — sympathetic arousal lifts her resistance **above normal**. *Resistance*: coping with the daily demands of caring for **several months** is the **resistance** stage — resistance stays above normal, sustained by cortisol, while her resources deplete. *Exhaustion*: becoming **chronically fatigued, run-down and frequently unwell** is the **exhaustion** stage — her resources are depleted, resistance falls **below normal** and she is vulnerable to illness.

Q11. In the **resistance** stage the body sustains above-normal resistance by continually releasing **cortisol**, drawing down its **resources**. If the stressor persists into the **exhaustion** stage, those **resources become depleted**, so the body can no longer maintain its defences and resistance falls **below normal**. At the same time, the **prolonged high cortisol suppresses the immune system**. With depleted resources and weakened immunity, the person is far **more vulnerable to illness** and stress-related disorders.

Q12. The statement is **incorrect**. During the **shock** sub-phase, the body reacts as if injured and its resistance to the stressor **drops below its normal level** — not above it. Resistance only **rises above normal** in the **counter-shock** sub-phase, once the sympathetic nervous system has mobilised the body for action.

Q13. (a) Region C is the **exhaustion** stage: resistance **falls, ending below the normal level** — the body's resources are depleted, so it can no longer sustain its defences. (b) Region A is the **alarm reaction**: the **dip below** the normal line is produced by the **shock** sub-phase (the body reacts as if injured, before it has mobilised); the following **rise above** the normal line is produced by the **counter-shock** sub-phase (sympathetic arousal). (c) Region B is the **resistance** stage, and the curve sitting **above** the normal line there represents the body **copied with the stressor at an elevated level of resistance** — a sustained, above-normal state of arousal held up by **cortisol** while the body's resources are gradually used up.

Q14. (a) A **strength** is that a **controlled animal experiment** lets the researcher **manipulate the stressor and hold other conditions constant**, so the physiological changes can be **measured objectively** and a consistent pattern established — giving the model a solid empirical basis. (b) A **limitation** is that **rats differ physiologically and cognitively from humans**, so a pattern seen in rats **may not generalise fully to people**. (c) Animal studies can only capture the **biological** side of stress; they cannot capture how a **person appraises** a stressor. This is the same gap that limits GAS itself — because the evidence (and the model built on it) is purely physiological, it **omits the psychological, cognitive dimension** of human stress.

Q15. GAS explains stress as a **fixed, non-specific biological sequence** — the same alarm– resistance–exhaustion pattern of physiological change for any stressor. A **psychological account** instead explains stress through the person’s **appraisal** — how **threatening or manageable** they *judge* the stressor to be. Because GAS assumes the **same biological response for everyone** and ignores appraisal, it **cannot explain individual differences**: it has no way to account for why one person is overwhelmed by an event that another person barely registers.

Q16. (a) “**Non-specific**” means that, according to GAS, the **same general physiological pattern** occurs **regardless of the type of stressor** — whether the stressor is heat, injury or an exam, the body runs through the same alarm– resistance–exhaustion sequence. (b) This is a **limitation** because later evidence suggests the stress response is **not entirely uniform** — it can **vary with the type of stressor** and with the **individual** (their appraisal, coping and physiology). Treating every response as the same **oversimplifies** the reality of stress.

Q17. (a) The **independent variable** is the **duration of exposure** to the stressor (**3 days vs 30 days**). The **dependent variables** are the rats’ **blood cortisol concentration** and the **degree of immune-tissue damage**. (b) Based on GAS, **Group 2 (30 days)** is more likely to show **depleted resources and immune damage**, because prolonged exposure moves the body out of the **resistance** stage and into the **exhaustion** stage, in which resources are spent and sustained cortisol has suppressed immune function. (c) The study used **rats**, which differ from humans, so the findings should be **generalised to humans with caution** — and, because it measures only physiology, it does not capture the **psychological appraisal** that shapes human stress.

Q18. According to **GAS**, stress is a **non-specific biological response** — the same physiological sequence for any stressor — and the model makes **no reference to how a person interprets** the situation. But here the two colleagues face the **same stressor** (the deadline) and respond **very differently**: one **appraises** it as an exciting **challenge**, the other as an overwhelming **threat**. This difference comes from their **cognitive appraisal**, which GAS does not include. So GAS, on its own, **cannot explain** why the same event produces energised coping in one colleague and dread in the other — a psychological model of appraisal is needed to account for it.

Q19. Model response (6 marks). Selye’s **General Adaptation Syndrome (GAS)** is a **biological model** that explains stress as a predictable **three-stage** physiological sequence: the **alarm reaction** (with a **shock** sub-phase, where resistance drops below normal, and a **counter-shock** sub-phase, where sympathetic arousal lifts resistance above normal), the **resistance** stage (resistance stays above normal, sustained by **cortisol**, while resources deplete), and the **exhaustion** stage (resources depleted, resistance falls below normal, the person becomes vulnerable to illness).

The model has real **strengths**. It **identifies a clear, predictable biological pattern**, giving a useful framework for understanding the body’s response to prolonged stress. It is **grounded in objective, measurable physiological evidence** gathered under **controlled experimental conditions**. And its exhaustion stage **explains the link between chronic stress and illness**, which has genuine clinical value.

However, GAS also has important **limitations**. It was developed largely from **animal (rat) studies**, so its findings **may not generalise fully to humans**. More fundamentally, it is a purely **biological** model that **ignores psychological and cognitive factors**, especially a person’s **subjective appraisal** of the stressor; because it treats the response as **non-specific** and the same for everyone, it **cannot explain individual differences** in how people respond to the same event.

Judgement: GAS is a **valuable and well-evidenced biological description** of how the body responds to a prolonged stressor, and it remains influential for that reason. But it is **not a complete explanation** of human stress, because it leaves out the **psychological, appraisal-based dimension** — which is why it is best regarded as **one part** of a fuller account of stress rather than the whole story. *(Full marks require: correct outline of the stages; at least two genuine strengths and two genuine limitations; and an explicit, justified judgement.)*

Chapter 4 Stress as a psychobiological process — 4D Lazarus and Folkman's Transactional Model of Stress and Coping

Theory

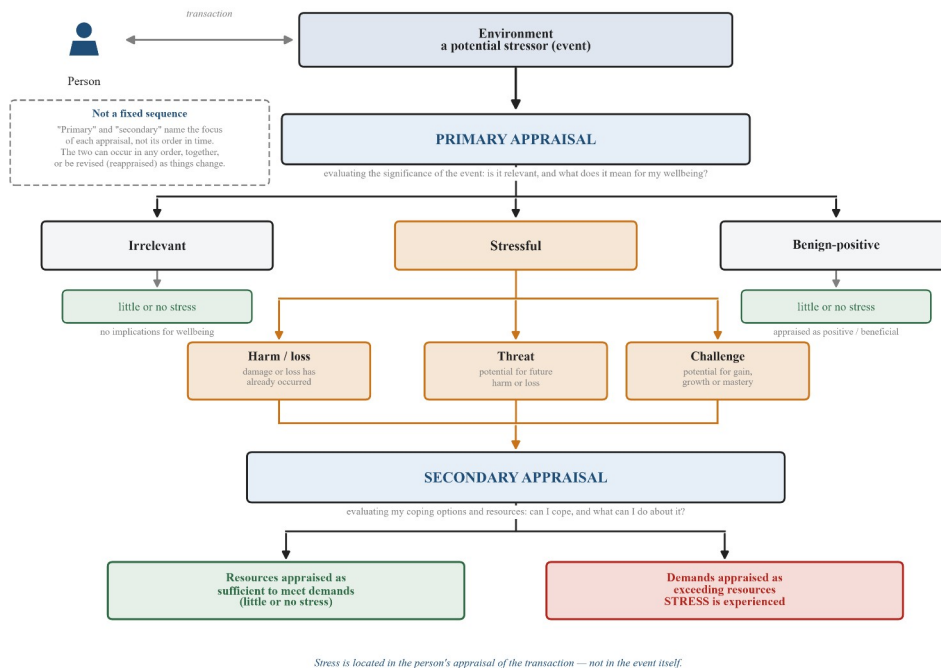
Lazarus and Folkman's Transactional Model of Stress and Coping is a **psychological** model of stress. Where a biological model such as Selye's General Adaptation Syndrome describes the body's **physiological** reaction to a stressor and treats the response as much the same for everyone, the Transactional Model locates stress in the **mind** of the person. It proposes that stress arises from a **transaction** — a two-way, ongoing relationship — between the **person** and their **environment**, and that whether an event becomes stressful depends on how the person **thinks about (appraises)** it. The one-line contrast to keep straight: **GAS is biological and ignores cognition; the Transactional Model is psychological and centres on the person's subjective appraisal.**

Appraisal is the mental process of evaluating a stimulus or situation. The model describes **two kinds** of appraisal — **primary appraisal** and **secondary appraisal**.

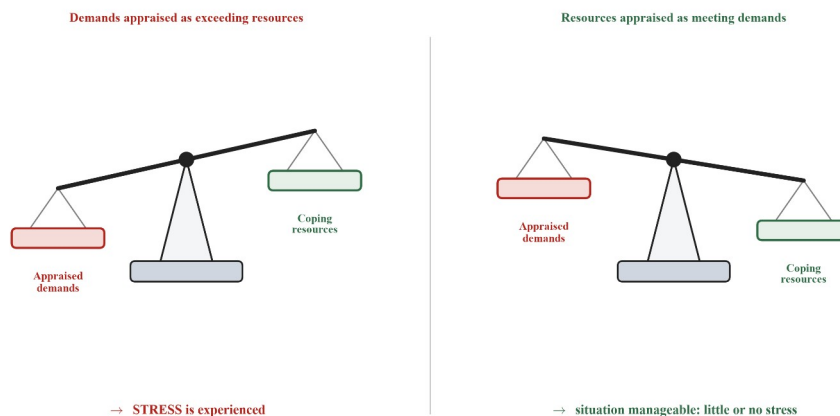
Primary appraisal is the person's judgement of **what the event means for their wellbeing** — “Is this relevant to me, and if so, is it good or bad for me?” A stimulus is judged to be one of three things:

- **Irrelevant** — the event has **no implications** for the person's wellbeing, so it is set aside (for example, hearing that rain is forecast for a city you are not visiting).
- **Benign-positive** — the event is appraised as **good or beneficial**, producing positive rather than stressful feelings (for example, being told class is cancelled).
- **Stressful** — the event is appraised as **threatening the person's wellbeing** in some way. A stressful primary appraisal is then further judged as one (or more) of:
 - **Harm/loss** — damage or loss has **already occurred** (an injury, a failed subject, the end of a relationship);
 - **Threat** — there is the **potential for future** harm or loss that has not happened yet (worrying that an upcoming exam might be failed);
 - **Challenge** — there is the **potential for gain, growth or mastery** — the demanding situation is viewed as an opportunity to be met (seeing that same exam as a chance to prove yourself).

Secondary appraisal is the person's evaluation of their **coping resources and options** — “Can I cope with this? What can I do about it, and do I have what I need to deal with it?” Here the person takes stock of what is available to them (their skills, time, energy, support from others, past experience) and judges whether these are **sufficient** to meet the demands of the situation. Secondary appraisal is about **appraising** one's ability to cope; the specific coping **strategies** a person then chooses are a separate matter, covered later in this chapter.



When is stress experienced? According to the model, **stress is experienced when the demands of the situation (appraised in primary appraisal) are judged to exceed the person's coping resources (appraised in secondary appraisal)**. If the person appraises their resources as **sufficient** to meet the demands, the situation is judged manageable and little or no stress results; if the demands are appraised as **outweighing** the resources, stress is experienced. Crucially, it is the person's **appraisal** of the demands and resources — not the objective size of the event — that matters.



Key features to keep straight.

- **Appraisal is subjective.** Because stress depends on how the individual *interprets* an event, the **same stimulus can be stressful for one person and not for another** — and can even be appraised differently by the *same* person at different times. Two students sitting the identical exam may appraise it as a threat and a challenge respectively, and so experience very different levels of stress.
- **“Primary” and “secondary” refer to the *focus* of each appraisal, not a fixed order in time.** Appraisals can occur in **any order, simultaneously**, or be **revised (reappraised)** as new information arrives or the situation changes — they are not rigid, one-after-the-other stages.
- **The relationship is two-way (a “transaction”).** The person acts on the environment and the environment acts back on the person; stress emerges from this ongoing **relationship**, not from the event alone. This is why the model is called *transactional*.

The explanatory power of the model — strengths and limitations. “*Explanatory power*” means **how well the model actually accounts for stress**. Listing strengths and limitations in the abstract is not enough: when a **scenario** is given, the evaluation must be **applied to that scenario** — say how well the model explains *that* person’s experience,

and name what it leaves unexplained. The application that comes up most often: because the model explains **psychological appraisal** and not the body's reaction, it has **low explanatory power** for anything **physiological** in a scenario (a racing heart, raised blood pressure, weakened immunity) and for responses that happen **without a conscious appraisal** — a **biological** model is needed for those.

Strengths:

- It accounts for the **psychological and subjective** nature of stress — it recognises that stress begins with how a person *thinks about* an event, not just how the body reacts.
- It **explains individual differences**: because appraisal is subjective, the model can explain why the same stressor stresses one person and not another, something a purely biological model cannot.
- It treats the person and the environment as a **two-way transaction**, capturing the idea that people actively interpret and respond to their circumstances rather than passively reacting.

Limitations:

- Appraisals are **difficult to measure or test objectively** — they are private mental processes, usually assessed through **subjective self-report**, which may be inaccurate or influenced by what the person is willing to reveal.
- The **appraisal processes overlap** and are not always **conscious or sequential**, which makes it hard to clearly separate primary from secondary appraisal in practice or to identify exactly when each occurs.
- It largely **ignores the biological/physiological** stress response — the bodily changes described by biological models — so it gives an incomplete picture of stress on its own.

Worked examples

Example 1 — scaffolded

Priya is told she must deliver a five-minute oral presentation to her class next week. She thinks, “This really matters — it counts towards my final mark and I could embarrass myself,” and then, “But I have a week to prepare, I’ve presented before, and my friend will help me rehearse.” Identify Priya’s primary and secondary appraisals, classify her primary appraisal, and state whether she is likely to experience high or low stress.

Working	Comment
First thought — “this matters... I could embarrass myself” — evaluates what the event means for her wellbeing → this is her primary appraisal .	Primary appraisal = judging the significance of the event <i>for me</i> .
She judges the presentation as stressful , and specifically as a threat (potential <i>future</i> harm — embarrassment that has not yet happened).	If stressful, classify as harm/loss, threat or challenge. “Could” embarrass = anticipated, so threat .
Second thought — “I have a week, I’ve done this before, a friend will help” — evaluates her coping resources and options → this is her secondary appraisal .	Secondary appraisal = “Can I cope? What resources do I have?”
Her appraised resources (time, experience, support) are judged sufficient to meet the appraised demands .	Stress depends on the balance of appraised demands vs appraised resources.
Because resources are appraised as meeting the demands, Priya is likely to experience relatively low stress .	$\text{Demands} \leq \text{resources}$ → situation judged manageable.

Example 2 — scaffolded

Tomas and Ruby are told the same news: each has been shortlisted for a promotion that requires a public interview in front of a panel. Tomas thinks, “This is my chance to show what I can do.” Ruby thinks, “I’m going to freeze and humiliate myself — I can’t handle this.” Explain, using primary and secondary appraisal, why the same event may be far more stressful for Ruby than for Tomas.

Working	Comment
The event is identical for both, so any difference in stress must come from their appraisals , not the event.	The model locates stress in appraisal, not in the stimulus.
Tomas's primary appraisal classifies the interview as a challenge (potential for gain/mastery); Ruby's classifies it as a threat (potential future harm/humiliation).	Same event, different <i>primary</i> appraisal — the subjective nature of the model.
In secondary appraisal , Tomas judges his resources as sufficient ("I can show what I can do"); Ruby judges hers as insufficient ("I can't handle this").	Secondary appraisal = evaluating coping resources relative to demands.
For Ruby, appraised demands exceed resources → stress ; for Tomas, resources meet demands → little stress.	Stress = appraised demands exceeding appraised resources.

Example 3 — unscaffolded

Mei is called to an unexpected meeting with her manager. Her heart races and her stomach tightens as she walks to the office. She thinks, "This must be about the complaint — I could lose my job," and then, "I have no idea how I would pay my rent if that happened." She remains highly stressed throughout the meeting. Evaluate the explanatory power of the Transactional Model of Stress and Coping for Mei's experience.

The model has **high explanatory power** for the **psychological** side of Mei's experience. Her thought that she could lose her job is a **primary appraisal** of the meeting as **stressful**, and specifically as a **threat** — the harm is anticipated, not yet done. "I have no idea how I would pay my rent" is a **secondary appraisal** in which she judges her **coping resources as insufficient**, so her appraised demands **exceed** her appraised resources and the model correctly predicts the high stress she reports. The model also explains why a colleague called to the same meeting expecting good news would appraise it as benign-positive and feel nothing — an **individual difference** a purely biological model cannot account for.

The model has **low explanatory power** for the rest of the scenario. Mei's **racing heart** and **tightened stomach** are **physiological** changes, and the Transactional Model explains **appraisal**, not the body's reaction — it says nothing about the sympathetic nervous system or the hormones that produce them, so a **biological** model such as Selye's General Adaptation Syndrome is needed there. Nor does it explain easily why her bodily response began **as she walked to the office**, before any deliberate appraisal had been made; and her appraisals are **private mental events** that can only be checked by **self-report**, so the explanation cannot be tested directly. ∴ the Transactional Model explains well *why the meeting was stressful for Mei* — how she interpreted it and how she judged her resources — but it has **low explanatory power** for her physical reaction, so a complete account of this scenario needs a biological model alongside it.

Example 4 — unscaffolded

Distinguish between Selye's General Adaptation Syndrome and Lazarus and Folkman's Transactional Model of Stress and Coping.

Selye's General Adaptation Syndrome is a **biological** model: it describes stress as a **physiological** response of the body that unfolds through set stages (alarm, resistance, exhaustion) and treats the response as broadly the **same for everyone**, regardless of how the person thinks. Lazarus and Folkman's Transactional Model, by contrast, is a **psychological** model: it explains stress in terms of the person's **subjective appraisal** of an event — a primary appraisal of what the event means for wellbeing, and a secondary appraisal of whether they can cope — so that the **same event can be stressful for one person and not another**. In short, GAS focuses on the **body and ignores cognition**, whereas the Transactional Model focuses on the **mind and the individual's interpretation** of the situation. ∴ the two models differ in whether they treat stress as a uniform biological response or as a subjective, cognitively appraised experience.

Practice questions

Scaffolded

- Q1.** Define each of the following in the context of the Transactional Model: (a) primary appraisal; (b) secondary appraisal; (c) a benign-positive appraisal.
- Q2.** Naomi's car breaks down on the way to an important job interview. She thinks, "This is a disaster — I'm going to miss the interview," and then, "But I could call a taxi or ask my neighbour for a lift." (a) Identify Naomi's primary appraisal and classify it as harm/loss, threat or challenge. (b) Identify Naomi's secondary appraisal. (c) State what determines, according to the model, whether Naomi experiences stress.
- Q3.** When an event is appraised as stressful in primary appraisal, it is further judged as one of three types. (a) Name the three types. (b) For each, state briefly what it means.
- Q4.** Ethan and Zara are given the same challenging research assignment. Ethan sees it as an exciting chance to explore a topic he loves; Zara is convinced she will fail it. (a) Classify each student's primary appraisal (harm/loss, threat, benign-positive or challenge). (b) Explain why the same assignment leads to different appraisals.
- Q5.** Diego's bicycle, which he uses to get to work, is stolen. (a) Classify Diego's likely primary appraisal of the theft. (b) Describe what Diego would evaluate during secondary appraisal. (c) Explain how secondary appraisal could influence whether Diego experiences ongoing stress.
- Q6.** The Transactional Model has both strengths and limitations as an explanation of stress. (a) State one strength of the model. (b) State one limitation of the model.

Un scaffolded

- Q7.** Distinguish between primary appraisal and secondary appraisal. (2 marks)
- Q8.** While bushwalking, Sofia notices dark storm clouds gathering and thinks she may be caught in dangerous weather far from shelter. She then considers that she has a map, a raincoat and enough daylight to reach the car park. (a) Classify Sofia's primary appraisal and justify your choice. (b) Identify her secondary appraisal. (c) Predict whether Sofia is likely to experience high or low stress, and justify your prediction using the model. (4 marks)
- Q9.** Distinguish between an **irrelevant** primary appraisal and a **benign-positive** primary appraisal, using an example of each, and state what the model predicts about the person's stress in each case. (3 marks)
- Q10.** Reuben's family moved house last month, which he found tiring but not especially stressful. This morning his printer jammed ten minutes before an assignment was due, and he felt intensely stressed. Using the Transactional Model, explain how a much smaller event can produce far more stress than a much larger one. Refer to both primary and secondary appraisal. (4 marks)
- Q11.** A student writes: "In the Transactional Model, primary appraisal always happens first and secondary appraisal always happens afterwards, and stress comes from how threatening the event actually is." Identify and correct **two** errors in this statement. (2 marks)
- Q12.** Distinguish between a **threat** appraisal and a **challenge** appraisal, using an example of a rock climber attempting a difficult climb. (3 marks)
- Q13.** Identify **one strength** of the Transactional Model and explain why it is considered a strength. (2 marks)
- Q14.** Identify **one limitation** of the Transactional Model and explain why it is considered a limitation. (2 marks)
- Q15. Research scenario.** A psychologist investigates whether the way a task is framed changes how stressful students find it. Fifty-two Year 11 students are randomly allocated to two groups. Group A is told an upcoming problem-solving task is "a chance to show what you can do" (a challenge framing); Group B is told the task "will be marked and compared with the rest of the class" (a threat framing). All students then rate how stressed they feel from 1 (not at all stressed) to 7 (extremely stressed). (a) Identify the independent variable and the dependent variable. (b) Identify the population the researcher is likely trying to draw a conclusion about. (c) The students were not told the real aim of the study until afterwards. Name the relevant ethical guideline and state what the researcher must do at the end of the study. (4 marks)

Q16. Research scenario. To study appraisal, a researcher asks students to complete a self-report questionnaire on the morning of a major exam, rating how threatening they find it and how well they think they can cope. (a) State whether primary appraisal, secondary appraisal, or both are being measured, and justify your answer with reference to the questionnaire. (b) Explain **one** limitation of measuring appraisals in this way, linking it to a limitation of the Transactional Model itself. (4 marks)

Q17. After being told she needs minor surgery, Harper first appraises the news as a serious threat and feels very stressed. After speaking with her surgeon and learning the procedure is routine and low-risk, she feels much calmer. Using the Transactional Model, explain what has occurred, referring to the idea that appraisals are not fixed. (3 marks)

Q18. Explain why the model is described as a *transactional* model of stress. (2 marks)

Q19. Extended response. Two weeks out from her final exams, Anika is told that her hours at her part-time job are being cut. She thinks, “I won’t be able to pay for my textbooks,” and can see no way to make up the money. Over the following fortnight she sleeps badly, her appetite drops, and a cold she catches lingers far longer than usual.

Evaluate the explanatory power of Lazarus and Folkman’s Transactional Model of Stress and Coping as an explanation of Anika’s stress. Refer to at least **two strengths** and **two limitations**, apply each to Anika’s situation, and reach an overall judgement. (6 marks)

Answers

Q1. (a) The person's judgement of what an event means for their wellbeing (irrelevant, benign-positive or stressful). (b) The person's evaluation of their coping resources and options ("can I cope?"). (c) An event appraised as good or beneficial for wellbeing. **Q2.** (a) Primary appraisal = "this is a disaster, I'll miss the interview"; classified as a **threat** (potential future harm). (b) Secondary appraisal = considering options/resources (call a taxi, ask the neighbour). (c) Whether she appraises her coping resources as sufficient to meet the demands — stress occurs if demands are appraised as exceeding resources. **Q3.** (a) Harm/loss, threat, challenge. (b) Harm/loss = damage/loss has already occurred; threat = potential for future harm/loss; challenge = potential for gain, growth or mastery. **Q4.** (a) Ethan = challenge; Zara = threat. (b) Appraisal is subjective, so the same event is interpreted differently by different people. **Q5.** (a) Harm/loss (the bike is already gone). (b) His coping resources and options — money to replace it, other transport, insurance, help from others. (c) If he appraises his resources as sufficient, stress is reduced; if insufficient, stress continues. **Q6.** (a) e.g. It explains individual differences in stress / accounts for the subjective, psychological nature of stress. (b) e.g. Appraisals are hard to measure objectively / it ignores the biological stress response. **Q7.** Primary appraisal judges what the event means for wellbeing (irrelevant/benign-positive/stressful); secondary appraisal evaluates whether the person can cope and what resources/options they have. **Q8.** (a) Stressful — a **threat** (potential future harm from the storm). (b) Evaluating her resources: map, raincoat, daylight to reach the car park. (c) Likely **low/manageable** stress, because she appraises her resources as sufficient to meet the demands. **Q9.** Irrelevant = the event has **no implications** for wellbeing (e.g. hearing that a sporting club you do not follow has changed coaches); benign-positive = the event **does** matter but is appraised as **good or beneficial** (e.g. being told a shift you had been dreading has been covered). Neither is appraised as threatening wellbeing, so the model predicts **little or no stress** in either case. **Q10.** Stress depends on the **appraisal**, not the objective size of the event. Primary appraisal: the jam is appraised as **stressful** — a **threat** (the deadline may be missed), the move as demanding but not threatening. Secondary appraisal: with ten minutes left Reuben appraises his resources as **insufficient**, whereas for the move he had weeks, help and a plan (**sufficient**). Stress results because appraised demands exceed appraised resources. **Q11.** Error 1: the appraisals do **not** occur in a fixed order — they can occur in any order, simultaneously, or be reappraised. Error 2: stress comes from the person's **appraisal**, not from how threatening the event "actually" is. **Q12.** Threat = the climb is appraised as potential **future harm** (falling/failing), producing stress; challenge = the climb is appraised as an **opportunity for mastery/gain**, producing a more positive state. Same climb, different appraisal. **Q13.** e.g. It explains individual differences — because appraisal is subjective, the model can explain why the same stressor affects people differently, which a biological model cannot. **Q14.** e.g. Appraisals are hard to measure objectively — they are private mental processes assessed by subjective self-report, which may be inaccurate. **Q15.** (a) IV = task framing (challenge vs threat); DV = self-reported stress rating (1–7). (b) The population = Year 11 students (the broader group the 52 students represent). (c) The guideline is the **use of deception**; the researcher must **debrief** participants (explain the true aim) afterwards. **Q16.** (a) Both — "how threatening" measures primary appraisal; "how well they can cope" measures secondary appraisal. (b) Self-report is subjective and may be inaccurate; this reflects the model's limitation that appraisals are hard to measure objectively. **Q17.** Harper's initial appraisal (threat → stress) changed after new information; she **reappraised** the situation as low-risk, showing appraisals are not fixed and can be revised. **Q18.** Because stress arises from a two-way **relationship (transaction)** between the person and the environment, not from the event alone. **Q19.** Model response given in the solutions.

Fully-worked solutions

Q1. (a) **Primary appraisal** is the person's judgement of **what an event means for their wellbeing** — whether it is **irrelevant, benign-positive**, or **stressful**. (b) **Secondary appraisal** is the person's **evaluation of their coping resources and options** — a judgement of whether they can cope with the situation and what they can do about it. (c) A **benign-positive** appraisal is a primary appraisal in which the event is judged to be **good or beneficial** for the person's wellbeing, producing positive rather than stressful feelings.

Q2. (a) Naomi's **primary appraisal** is her thought that the breakdown is "a disaster" and she will miss the interview — she judges the event as **stressful**, and specifically a **threat** (the harm — missing the interview — is anticipated, not yet certain). (b) Her **secondary appraisal** is her evaluation of her **coping options and resources**: calling a taxi or asking her neighbour for a lift. (c) According to the model, whether Naomi experiences stress depends on the **balance between her appraised demands and her appraised coping resources** — she will experience stress if she appraises

the demands as **exceeding** her resources, and little stress if she appraises her resources (taxi, lift) as **sufficient** to meet them.

Q3. (a) The three types are **harm/loss**, **threat** and **challenge**. (b) **Harm/loss** means damage or loss has **already occurred** (e.g. an injury or a failed subject); **threat** means there is **potential for future** harm or loss that has not yet happened (e.g. worry about an upcoming exam); and **challenge** means the demanding situation is appraised as an **opportunity for gain, growth or mastery**.

Q4. (a) **Ethan's** primary appraisal is a **challenge** (he sees the assignment as an exciting opportunity), while **Zara's** is a **threat** (she anticipates failing — future harm). (b) The assignment is **identical**, so the difference lies in the students' **subjective appraisals**. Because appraisal depends on how each person interprets the event — shaped by their confidence, past experience and how they judge their resources — the same assignment can be appraised as a challenge by one student and a threat by another.

Q5. (a) Diego's likely primary appraisal is **stressful** — **harm/loss**, because the bike is **already gone** (a loss that has already occurred). (b) During **secondary appraisal**, Diego evaluates his **coping resources and options** — for example, whether he has money to replace the bike, access to other transport, insurance, or people who could help. (c) If Diego appraises these resources as **sufficient** to deal with the loss (e.g. he can easily afford another bike or catch a bus), his ongoing stress will be **low**; if he appraises his resources as **insufficient** (no money, no other way to get to work), the appraised demands exceed his resources and he is likely to experience **continuing stress**.

Q6. (a) One strength is that the model **accounts for individual differences / the subjective nature of stress** — it explains why the same event affects different people differently. (b) One limitation is that **appraisals are difficult to measure objectively** (they rely on subjective self-report), or that the model **ignores the biological stress response**. (Either strength/limitation pairing is acceptable.)

Q7. **Primary appraisal** is the person's judgement of **what an event means for their wellbeing** — whether it is irrelevant, benign-positive or stressful (and, if stressful, harm/loss, threat or challenge). **Secondary appraisal** is the person's evaluation of **whether they can cope** — a judgement of their coping **resources and options** relative to the demands of the situation. (Both sides contrasted for the two marks.)

Q8. (a) Sofia's primary appraisal is **stressful**, and specifically a **threat**, because the storm represents **potential future harm** (danger from the weather) that has **not yet occurred**. (b) Her **secondary appraisal** is her evaluation of her **coping resources**: she has a **map, a raincoat and enough daylight** to reach the car park safely. (c) Sofia is likely to experience **relatively low (manageable) stress**, because although she appraises the situation as a threat, in secondary appraisal she judges her resources as **sufficient to meet the demands** — and the model states that stress is experienced only when appraised demands **exceed** appraised resources.

Q9. Both are **primary appraisals** — judgements of what an event means for the person's wellbeing — and neither is a **stressful** appraisal, but they differ in *why*. An **irrelevant** appraisal is one in which the event has **no implications** for the person's wellbeing at all: nothing is at stake, so the event is simply set aside — for example, hearing that a sporting club the person does not follow has changed coaches. A **benign-positive** appraisal is one in which the event **does** have implications for wellbeing, but they are judged **good or beneficial**, producing positive rather than stressful feelings — for example, being told that a shift the person had been dreading has been covered by a colleague. In **neither** case is the event appraised as threatening wellbeing, so in neither case can appraised demands exceed appraised coping resources: the model predicts **little or no stress** in both, and neither requires the person to weigh up coping options as a stressful appraisal would.

Q10. According to the Transactional Model, stress depends on the person's **appraisal** of an event, **not on the objective size** of the event — so a small event can be appraised as far more stressful than a large one. In **primary appraisal**, Reuben judges the printer jam as **stressful** — **specifically a threat**, because it carries **potential future harm** (a missed deadline and lost marks) that has not yet occurred; the house move, although objectively far bigger, was appraised as demanding but **not threatening** to his wellbeing. In **secondary appraisal**, he evaluates his **coping resources and options** in each case: with **ten minutes** left he has no time, no second printer and no chance to ask for an extension, so his resources are appraised as **insufficient**, whereas for the move he had weeks of notice, his family's help and a plan, so his resources were appraised as **sufficient** to meet the demands. Because the model states that stress is experienced when **appraised demands exceed appraised resources**, the printer jam — small, sudden and

with no resources available to meet it — produces **intense stress**, while the much larger move, met with ample appraised resources, produces little.

Q11. Error 1: primary and secondary appraisal do **not** occur in a fixed order — “primary” and “secondary” refer to the **focus** of each appraisal, not their timing; they can occur in **any order, simultaneously, or be reappraised**. **Error 2:** according to the model, stress comes from the person’s **subjective appraisal** of the event, **not** from how threatening the event “actually” is — the same event can be stressful for one person and not another.

Q12. A **threat** appraisal judges the climb as a source of **potential future harm** — the climber focuses on the possibility of **falling or failing**, which produces stress and negative emotion. A **challenge** appraisal judges the same climb as an **opportunity for gain, growth or mastery** — the climber focuses on the chance to **test and improve their skills**, producing a more positive, motivated state. The **climb is the same**; the difference lies in whether it is appraised as potential harm (threat) or potential gain (challenge).

Q13. One **strength** is that the model **explains individual differences in stress**. Because stress depends on **subjective appraisal**, the model can explain why the **same stressor** produces stress in one person but not another — something a purely **biological** model (which treats the stress response as the same for everyone) cannot do. This makes it a strong account of the **psychological** side of stress.

Q14. One **limitation** is that **appraisals are difficult to measure objectively**. Appraisals are **private mental processes** that cannot be observed directly, so researchers must rely on **subjective self-report**, which may be **inaccurate**, incomplete, or affected by what the person is willing or able to report. This makes the model’s central process hard to **test scientifically**. (Alternatively: because the appraisals **overlap and are not always conscious or sequential**, they are hard to separate; or the model **ignores the biological stress response**, giving an incomplete picture.)

Q15. (a) The **independent variable** is the **framing of the task** (a challenge framing vs a threat framing). The **dependent variable** is the **self-reported stress rating** (the score from 1 to 7). (b) The **population** is the broader group the researcher wants to draw a conclusion about — **Year 11 students** — of whom the 52 participants are a **sample**. (c) The relevant **ethical guideline** is the **use of deception**: the real aim was deliberately withheld, which is permitted only because telling the students the aim would have changed the very appraisals being measured. Use of deception must **always** be followed by the guideline of **debriefing**, so at the end of the study the researcher must inform all participants of the true aim, the results and the conclusions, correct any misunderstanding, and offer support so that participants leave in the state they arrived in. (Note that *deception* and *debriefing* are ethical **guidelines** — procedures — not ethical **concepts**; the concepts they serve here are **respect** for the participants’ autonomy and **non-maleficence**.)

Q16. (a) **Both** appraisals are being measured. Rating “**how threatening**” the exam is measures **primary appraisal** (the judgement of what the event means for wellbeing), while rating “**how well they think they can cope**” measures **secondary appraisal** (the evaluation of coping resources). (b) One limitation is that the questionnaire relies on **subjective self-report**: students may not accurately know or honestly report their own appraisals, and appraisals can **change** from moment to moment. This directly reflects a limitation of the **Transactional Model itself** — that appraisals are **private mental processes that are hard to measure objectively**, which makes the model difficult to test scientifically.

Q17. Harper’s response shows that appraisals are **not fixed** — they can be **reappraised** as new information arrives. Initially her **primary appraisal** of the surgery was a **threat** (potential future harm), and she experienced stress. After speaking with her surgeon, **new information** (that the procedure is routine and low-risk) led her to **reappraise** the situation — both re-evaluating the threat and judging her ability to cope as adequate — so her appraised demands no longer exceeded her resources and her stress fell. This illustrates the model’s feature that appraisals are **ongoing and revisable**, not one-off judgements.

Q18. The model is called *transactional* because it explains stress as arising from a **two-way relationship (a “transaction”)** between the person and their environment: the person appraises and acts on the environment, and the environment acts back on the person. Stress is located in this ongoing **relationship** — in how the person appraises the demands of the environment against their own resources — rather than in the **event alone**.

Q19. Model response (6 marks). The Transactional Model has **high explanatory power** for part of Anika’s experience and **low explanatory power** for the rest of it.

Strengths, applied to Anika. First, the model captures the **psychological and subjective** nature of stress, and this is exactly what Anika's stress consists of. Losing hours is not in itself harmful; what makes it stressful is her **primary appraisal** of it as a **threat** — “I won't be able to pay for my textbooks” is anticipated future harm — together with her **secondary appraisal** that she can **see no way** to make up the money, i.e. her coping resources are **insufficient**. The model therefore predicts precisely what happens: appraised demands **exceed** appraised resources, so stress is experienced. Second, the model **explains individual differences**, which is needed here: a classmate whose hours were cut in the same week, but who has savings and no textbooks left to buy, would appraise the identical event as **irrelevant or benign-positive** and feel no stress at all. A purely biological model, which treats the stress response as much the same in everyone, cannot explain why the same event flattens Anika and not her classmate. The model also frames her situation as an ongoing **two-way transaction** — the timing of the cut two weeks before exams, and Anika's own attempts to act on the problem, both feed back into how she appraises it.

Limitations, applied to Anika. However, the model's central process is **difficult to measure or test objectively**. Everything the model relies on in this scenario — that Anika appraised the news as a threat and her resources as inadequate — is a **private mental event** that can only be reached through her **self-report**, which may be inaccurate or incomplete; there is no way to verify the appraisal that is supposed to be doing the explanatory work. Second, and more seriously here, the model **largely ignores the biological/physiological** stress response, and much of what happens to Anika **is** physiological: her **disturbed sleep, loss of appetite** and especially the **cold that lingers** (suggesting a suppressed immune system under prolonged stress) are bodily changes the Transactional Model does not explain at all. For those, a **biological** model such as Selye's General Adaptation Syndrome — with its account of prolonged resistance depleting resources and leaving the body vulnerable to illness — is required. A related limitation is that the appraisals themselves **overlap and are not always conscious or sequential**, so it is hard to say cleanly when Anika's primary appraisal ended and her secondary appraisal began.

Judgement: Overall, the Transactional Model has **high explanatory power for why the news was stressful for Anika** — it explains her interpretation, her judgement of her resources, and why another student would have been unaffected — but **low explanatory power for her physical symptoms**. It is therefore a strong but **partial** explanation of this scenario, and is **most complete when used alongside a biological model** rather than on its own. *(Full marks require: at least two clearly explained strengths and two clearly explained limitations, each applied to Anika's situation rather than stated in the abstract, and an explicit overall judgement.)*

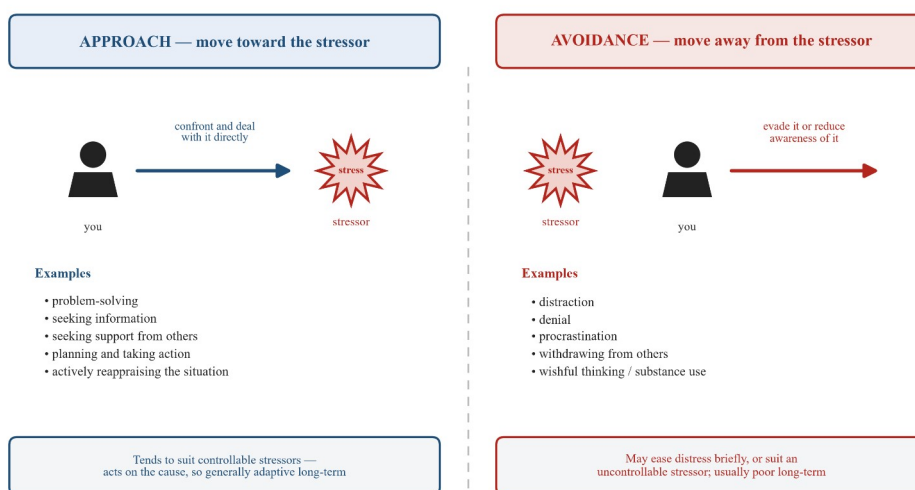
Chapter 4 Stress as a psychobiological process — 4E Coping strategies and coping flexibility

Theory

Coping is the process of managing, reducing or tolerating the demands created by a stressor. It refers to a person's **conscious, effortful attempts** to deal with the stress they are experiencing — whether by acting on the **stressor itself** or on their **emotional reaction** to it. People cope not only to reduce stress but also to protect and improve their **mental wellbeing**. (In the transactional model, **secondary appraisal** is the stage at which a person evaluates their coping options and resources; coping strategies are what they then put into action.)

Approach and avoidance. Coping strategies can be grouped by whether they move a person **toward** the stressor or **away** from it.

- **Approach strategies** confront the stressor and deal with it directly. The person engages with the source of the stress and takes active steps to manage it — for example, **seeking information, seeking help or support, problem-solving, planning and taking action**, or actively **reappraising** the situation. Because they act on the cause, approach strategies are generally the more **adaptive in the long term**: the stressor is more likely to be reduced or resolved.
- **Avoidance strategies** evade the stressor or reduce a person's **awareness** of it. Rather than engaging with the source of the stress, the person turns away from it — for example, through **denial, distraction, procrastination, withdrawing** from others, **wishful thinking**, or **using alcohol or other substances** to numb the feeling. Avoidance can lower distress **in the short term**, but because the stressor itself is not dealt with it is usually **maladaptive in the long term**: the problem persists or worsens and can accumulate.



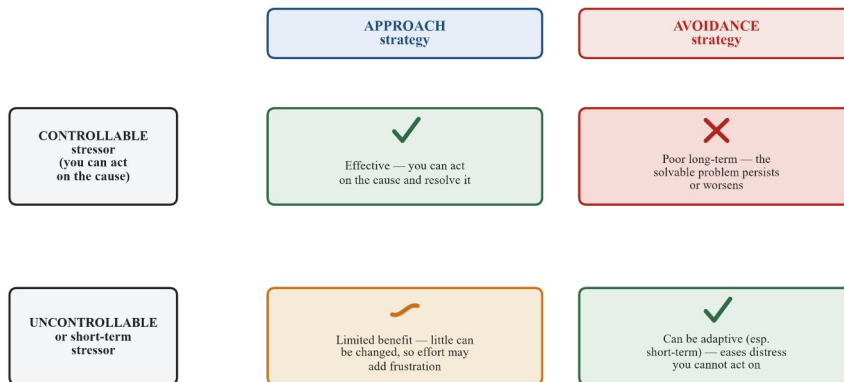
Direction, not comfort. What makes a strategy an **approach** or an **avoidance** strategy is whether the person **engages with** the stressor or **turns away** from it — not whether the strategy feels pleasant, and not whether the stressor is removed straight away. Phoning a friend to talk about a stressful situation and how it is affecting you is **seeking support**, so it is an **approach** strategy, even though nothing about the situation has changed yet. Turning on the television to stop thinking about that same situation directs attention **away** from it, so it is **avoidance**.

Context-specific effectiveness. No coping strategy is effective in every situation; a strategy's usefulness depends on the **context** — in particular, on whether the stressor is **controllable** and on the **time frame** involved.

- **Approach** strategies tend to be effective for **controllable** stressors, where there is something the person can actually do to change the situation (e.g. a looming assignment they can start work on).
- **Avoidance** strategies can be **adaptive in the short term**, or when a stressor is genuinely **uncontrollable** — repeatedly confronting a situation that cannot be changed, or that will resolve on its own, may only increase distress without any benefit (e.g. keeping yourself busy the evening before a decision that is already out of your hands is announced).

The **same** strategy can therefore be effective in one context and ineffective in another. This is what is meant by the **context-specific effectiveness** of a coping strategy: its value is judged *relative to the situation*, not as a fixed “good” or “bad”.

Context-specific effectiveness — the same strategy, different contexts



A strategy is not simply “good” or “bad” — its effectiveness depends on the context.

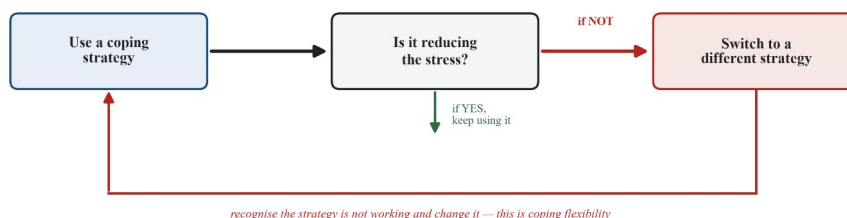
Why avoidance can help now but harm later. In the **short term**, avoidance can provide relief — it reduces overwhelming emotion and gives a person respite, which is useful when nothing can be done immediately. In the **long term**, however, avoiding a stressor that *could* be dealt with means the stressor is never addressed: it persists or grows, opportunities to act are missed, and avoidance can create *additional* problems (for example, procrastination leaving less time, or substance use creating its own harm). This is why avoidance is often **adaptive briefly but maladaptive if relied on**.

Coping flexibility. Coping flexibility is the ability to effectively **modify or adjust** one’s coping strategies to suit the **changing demands** of a stressful situation. A person with high coping flexibility **monitors** whether their current strategy is working and — if it is not — is willing and able to **abandon it and try a different one**. It therefore involves two abilities:

1. **evaluating** whether the strategy being used is actually reducing the stress; and
2. if it is not, **replacing** it with a more suitable strategy.

Coping flexibility matters because contexts **change**: a strategy that suited a stressor at the start may stop working as the situation develops. People with **high** coping flexibility tend to show **better adjustment and wellbeing**, because they match their coping to the situation rather than applying the same response rigidly. People with **low** coping flexibility keep using one strategy regardless of whether it is helping, which is associated with **poorer adjustment**.

Coping flexibility — adjusting the strategy to fit the changing demands



How to analyse a coping scenario. (1) Decide the **direction** of each strategy: does the person engage with the stressor (**approach**) or move away from it (**avoidance**)? (2) Judge its **context-specific effectiveness**: is the stressor **controllable**, and is the strategy suited to that context and time frame? (3) Where the situation changes, ask whether the person shows **coping flexibility** — do they recognise a strategy is not working and **switch** to a better one?

Worked examples

Example 1 — scaffolded

Priya has a major oral presentation in two weeks. In response she: (i) books a practice session with her teacher; (ii) writes herself a preparation timetable; (iii) spends several evenings binge-watching television to keep her mind off it; and (iv) tells herself the presentation will probably be cancelled. Classify each of Priya's four responses as an **approach** or an **avoidance** strategy, and justify each classification.

Working	Comment
(i) Booking a practice session engages with the stressor and takes direct action → approach .	Approach = moving <i>toward</i> the stressor to deal with it.
(ii) Writing a preparation timetable is planning and taking action on the task → approach .	Planning is a classic approach strategy — it acts on the cause.
(iii) Binge-watching television to keep her mind off it reduces her awareness of the stressor → avoidance (distraction).	Distraction turns attention <i>away</i> from the stressor.
(iv) Telling herself it will be cancelled denies the reality of the stressor → avoidance (denial/wishful thinking).	Denial evades the stressor rather than dealing with it.

Example 2 — scaffolded

Marcus uses **distraction** (an avoidance strategy) in two different situations: (a) during the five hours he is stuck at an airport because a storm has grounded his flight home; and (b) instead of starting a large assignment that is due next week. Evaluate the **context-specific effectiveness** of distraction in each situation.

Working	Comment
(a) The grounded flight is uncontrollable and short-lived — the weather is beyond Marcus's influence and the delay will end on its own. So distraction here is adaptive : listening to music occupies the wait and keeps his frustration manageable until the flight is called.	Identify controllability and time frame first. Avoidance can be effective for a genuinely uncontrollable, short-term stressor.
(b) The assignment is controllable — Marcus <i>can</i> act on it, and there is still time. So distraction here is ineffective / maladaptive : the solvable problem persists, time is lost, and stress will build as the deadline nears. The contrast shows the strategy's effectiveness is context-specific — it depends on the situation, not on the strategy alone.	Again, judge controllability. The <i>same</i> strategy is poorly suited to a controllable stressor. This is the key idea being assessed.

Example 3 — unscaffolded

Define **coping flexibility** and, using an example, explain how a student with **high** coping flexibility would differ from one with **low** coping flexibility when a coping strategy stops working.

Coping flexibility is the ability to modify or adjust one's coping strategies to suit the changing demands of a situation — recognising when a strategy is not working and switching to a more suitable one. Suppose two students are preparing for exams by re-reading their notes, and both find that re-reading is not helping them remember. The student with **high** coping flexibility notices that the strategy is not reducing their stress or improving their recall, abandons it, and switches to a different approach strategy such as practice questions or asking the teacher for help. The student with **low** coping flexibility keeps re-reading the notes rigidly, even though it is not working, and grows more stressed as the exam approaches. ∴ high coping flexibility involves monitoring and adjusting the strategy to fit the changing demands, and is associated with better adjustment, whereas low coping flexibility means persisting with an ineffective strategy.

Example 4 — unscaffolded

Evaluate the claim: “Avoidance coping is always a bad strategy.”

The claim is **too absolute**. It is true that avoidance is usually **maladaptive in the long term**, especially for a **controllable** stressor: because the stressor is never dealt with, the problem persists or worsens, opportunities to act are missed, and avoidance can create further problems (such as procrastination or substance use). However, avoidance is **not always** bad. In the **short term**, or when a stressor is genuinely **uncontrollable**, avoidance can be **adaptive** — it reduces overwhelming distress that the person cannot act on anyway, providing respite until the situation changes or passes. Its value is therefore **context-specific**. ∴ avoidance is not “always” bad; it can be adaptive briefly or for uncontrollable stressors, but is maladaptive when relied on for a stressor that could be confronted and resolved.

Practice questions

Scaffolded

- Q1.** Define each of the following: (a) coping; (b) an approach coping strategy; (c) an avoidance coping strategy.
- Q2.** State whether each of the following is an **approach** or an **avoidance** strategy: (a) making a detailed study plan; (b) refusing to think about a problem and hoping it goes away; (c) asking a school counsellor for advice.
- Q3.** Regarding the context-specific effectiveness of coping strategies: (a) explain what “context-specific effectiveness” means; (b) name a type of stressor for which an approach strategy is well-suited, and say why; (c) describe a situation in which an avoidance strategy could be adaptive, and say why.
- Q4.** Regarding coping flexibility: (a) define coping flexibility; (b) state the two abilities it involves; (c) state whether **high** or **low** coping flexibility is associated with better adjustment.
- Q5.** An avoidance strategy such as distraction is used to cope with an approaching stressful event. (a) State one **short-term** benefit of the strategy. (b) State one **long-term** cost if the stressor is controllable. (c) Explain why the long-term cost occurs.
- Q6.** Dev feels overwhelmed by a large project and copes by repeatedly cleaning his room to avoid starting it. Weeks pass and the project is still not begun. (a) Classify Dev’s strategy as approach or avoidance. (b) Explain whether this strategy is well-suited to the context. (c) Describe what Dev would do differently if he had high coping flexibility.

Unscaffolded

- Q7.** Distinguish between approach and avoidance coping strategies. (2 marks)
- Q8.** Explain why approach strategies are generally more adaptive in the long term than avoidance strategies. (2 marks)
- Q9.** A student writes: “Talking to a friend about how upset you are is an avoidance strategy, because it does not fix the problem.” Identify and correct the error in this statement. (2 marks)
- Q10.** Facing a stressful move to a new city, Aisha (i) researches the new area and organises her belongings, and (ii) spends hours scrolling social media to avoid thinking about the move. Classify each response as approach or avoidance, and evaluate which is likely to be more effective for coping with the move. (4 marks)
- Q11.** Explain what is meant by the *context-specific effectiveness* of a coping strategy, using an example of one strategy that is effective in one context but not another. (3 marks)
- Q12.** Define coping flexibility and explain why it is associated with better adjustment to stress. (3 marks)
- Q13.** Explain how an avoidance strategy can be adaptive in the short term but maladaptive in the long term. (4 marks)
- Q14.** Whenever Noah is stressed about anything — an argument, an exam, a deadline — he copes in the same way: he plays video games until he feels better. Identify the level of coping flexibility Noah is showing and explain how this could affect his adjustment to a stressor that video games do not resolve. (3 marks)

Q15. After a much-loved pet dies, Leila copes in the first few days by keeping busy and distracting herself rather than dwelling on the loss. A friend tells her she is “coping the wrong way”. With reference to context-specific effectiveness, explain why Leila’s strategy may in fact be appropriate at this time. (3 marks)

Q16. Research scenario. A health psychologist investigated whether the type of coping strategy university students habitually use is related to their psychological wellbeing. Eighty first-year students at one university completed a questionnaire that classified each of them as a predominantly **approach** coper or a predominantly **avoidance** coper, and then completed a standardised wellbeing scale (higher score = greater wellbeing, out of 60).

Coping style	Mean wellbeing score	Standard deviation
Approach copers	44.6	7.2
Avoidance copers	31.8	9.5

- (a) Identify the independent variable and the dependent variable.
- (b) Identify the population from which the sample was drawn.
- (c) Write a directional (one-tailed) hypothesis for this investigation.
- (d) With reference to the data, state the relationship the results show.
- (e) Name one ethical consideration the psychologist should address, given the topic. (6 marks)

Q17. Research scenario. In a workplace study, 60 employees were assessed and grouped as having **high** or **low** coping flexibility. During a demanding three-week project, each employee’s mean self-reported stress was recorded.

Group	Mean stress rating (0–10)	Standard deviation
High coping flexibility	4.1	1.3
Low coping flexibility	6.8	1.6

- (a) Identify the independent variable and the dependent variable.
- (b) The researchers did **not** randomly allocate employees to the two groups. Explain why this means they cannot conclude that coping flexibility *caused* the difference in stress.
- (c) Write a directional hypothesis for this investigation.
- (d) Suggest one reason why high coping flexibility might reduce stress across a changing project. (5 marks)

Q18. A student with a heavy workload feels anxious and, over one week, cancels social plans, stops exercising, and avoids opening the subject’s online page. Recommend **two** approach strategies the student could use instead, and explain how coping flexibility would help them manage the workload. (4 marks)

Q19. Extended response. Jordan tore a ligament at football training and has been told he will need about four months of rehabilitation, which means missing the rest of the season. Referring to **approach and avoidance strategies**, the **context-specific effectiveness** of a strategy, and **coping flexibility**, discuss how Jordan might cope across the four months of his recovery. (6 marks)

Answers

Q1. (a) The conscious, effortful process of managing, reducing or tolerating the demands of a stressor. (b) A strategy that confronts the stressor and deals with it directly. (c) A strategy that evades the stressor or reduces awareness of it.

Q2. (a) Approach. (b) Avoidance. (c) Approach. **Q3.** (a) A strategy's effectiveness depends on the situation, so no strategy is effective in every context. (b) A controllable stressor (e.g. an assignment), because approach acts on a cause the person can change. (c) A genuinely uncontrollable/short-term stressor (e.g. the night before a team list the selectors have already decided is posted), because avoidance eases distress no action can reduce.

Q4. (a) The ability to modify/adjust coping strategies to suit the changing demands of a situation. (b) Evaluating whether the current strategy is working, and replacing it if it is not. (c) High. **Q5.** (a) It eases distress/provides short-term relief. (b) The (solvable) stressor is not dealt with and persists or worsens. (c) Because avoidance does not act on the cause, so a controllable problem remains unresolved and accumulates.

Q6. (a) Avoidance. (b) Poorly suited — the project is controllable, so avoidance leaves it unresolved and stress builds. (c) He would notice the strategy is not working and switch to an approach strategy (e.g. making a plan and starting the project).

Q7. Approach strategies confront and deal directly with the stressor; avoidance strategies evade the stressor or reduce awareness of it.

Q8. Because they act on the cause, so the stressor is more likely to be reduced or resolved, whereas avoidance leaves it unaddressed.

Q9. Error: the direction of a strategy is not decided by whether the problem is fixed. Talking to a friend about the stressor is *seeking support* — it engages with the source of the stress — so it is an **approach** strategy.

Q10. (i) Approach; (ii) avoidance. The approach strategy is likely more effective because the move is controllable, so acting on it resolves demands; the avoidance strategy leaves the move unprepared.

Q11. It means a strategy's usefulness depends on the situation; e.g. distraction is adaptive while waiting for a decision the person cannot influence, but maladaptive when used to put off a solvable assignment.

Q12. The ability to adjust coping strategies to changing demands; it is linked to better adjustment because the person matches coping to the situation rather than persisting rigidly with what is not working.

Q13. Short term it reduces overwhelming distress/provides respite; long term the stressor is not dealt with, so it persists or worsens and can create further problems.

Q14. Low coping flexibility. If gaming does not resolve the stressor, persisting with the same strategy leaves the stressor unaddressed and worsens his adjustment.

Q15. The loss is uncontrollable and the stress is acute; distraction in the first few days eases distress no action of hers can relieve, so it is contextually appropriate (short-term, uncontrollable).

Q16. (a) IV = coping style (approach vs avoidance); DV = wellbeing score. (b) First-year university students (at that university). (c) See solutions. (d) Approach copers reported higher mean wellbeing (44.6) than avoidance copers (31.8). (e) Confidentiality of results / informed consent / access to support after a potentially sensitive questionnaire.

Q17. (a) IV = level of coping flexibility (high vs low); DV = self-reported stress rating. (b) Without random allocation the groups may differ on other (participant) variables, so causation cannot be inferred. (c) See solutions. (d) High flexibility lets employees switch strategies as project demands change, so stress is better managed.

Q18. Two approach strategies (e.g. making a study timetable; seeking help from the teacher) + coping flexibility lets the student monitor and switch strategies as the workload changes. (Model in solutions.)

Q19. Model response given in the solutions.

Fully-worked solutions

Q1. (a) **Coping** is the conscious, effortful **process of managing, reducing or tolerating the demands** created by a stressor (by acting on the stressor or on one's emotional response). (b) An **approach** coping strategy is one that **confronts the stressor and deals with it directly** — the person engages with the source of the stress (e.g. problem-solving, seeking support). (c) An **avoidance** coping strategy is one that **evades the stressor or reduces awareness of it** — the person turns away from the source of the stress (e.g. denial, distraction).

Q2. (a) **Approach** — making a study plan is planning/taking action on the stressor. (b) **Avoidance** — refusing to think about the problem reduces awareness of it. (c) **Approach** — asking a counsellor for advice engages with the problem by seeking support/information.

Q3. (a) **Context-specific effectiveness** means that a coping strategy's usefulness **depends on the situation** — the same strategy can be effective in one context and ineffective in another, so no strategy is universally "good" or "bad". (b) An approach strategy suits a **controllable** stressor, such as an upcoming assignment, because approach **acts on a cause the person can actually change**, so the stressor can be reduced or resolved. (c) An avoidance strategy could be adaptive when the stressor is **uncontrollable and/or short-term**, such as the night before a team list the selectors have already decided is posted: **distraction occupies the person's attention and eases distress that no action of theirs can reduce**, until the news arrives.

- Q4.** (a) **Coping flexibility** is the ability to **modify or adjust** one's coping strategies to suit the **changing demands** of a stressful situation. (b) It involves (1) **evaluating** whether the current strategy is reducing the stress, and (2) if it is not, **replacing** it with a more suitable strategy. (c) **High** coping flexibility is associated with better adjustment.
- Q5.** (a) In the **short term** the avoidance strategy **eases distress and provides relief/respite** from the stressor. (b) In the **long term**, if the stressor is **controllable**, it is **left unresolved** and **persists or worsens** (and time to act is lost). (c) This occurs because avoidance **does not act on the cause** of the stress: the person's attention is diverted, but the underlying (solvable) problem remains and can accumulate further problems.
- Q6.** (a) It is an **avoidance** strategy — cleaning is used to avoid engaging with the project. (b) It is **poorly suited to the context**: the project is a **controllable** stressor, so avoidance leaves it unresolved while the deadline approaches and stress builds; an approach strategy would fit better. (c) With **high coping flexibility**, Dev would **notice that avoidance is not reducing his stress** and **switch to an approach strategy** — for example, breaking the project into steps, making a plan and starting the first task.
- Q7.** **Approach** strategies **confront the stressor and deal with it directly**, engaging with the source of the stress (e.g. problem-solving). **Avoidance** strategies **evade the stressor or reduce awareness of it**, turning away from the source (e.g. distraction). The key contrast is engaging *with* versus turning *away from* the stressor.
- Q8.** Approach strategies are generally more adaptive in the long term because they **act on the cause of the stress**, so the stressor is **more likely to be reduced or resolved**. Avoidance strategies do not deal with the stressor, so it **remains unaddressed** and can persist or worsen over time.
- Q9.** The statement is **incorrect**. Whether a strategy is **approach** or **avoidance** depends on the **direction** of the coping effort — whether the person **engages with the stressor** or **turns away from it** — and not on whether the stressor is removed straight away. Talking to a friend about how the situation is affecting you is **seeking support**, which engages with the source of the stress, so it is an **approach** strategy. (Ringing the same friend in order to talk about anything *but* the stressor *would* be avoidance, because attention is being directed **away** from it.)
- Q10.** (i) Researching the area and organising belongings is an **approach** strategy — Aisha engages with the move and takes action on it. (ii) Scrolling social media to avoid thinking about the move is an **avoidance** strategy (distraction). The **approach** strategy is likely to be **more effective** for coping with the move because the move is a **controllable** stressor: preparing for it **acts on the demands** and reduces them, so the situation becomes more manageable. The avoidance strategy may briefly ease anxiety but leaves the move **unprepared**, so the stress returns and can build.
- Q11.** The **context-specific effectiveness** of a coping strategy means that its usefulness **depends on the situation**, so a strategy can be effective in one context and ineffective in another. For example, **distraction** is **adaptive** when used the night before a team list the selectors have already decided is posted (an uncontrollable, short-term stressor), because it eases distress that no action of the person's own can reduce; but the **same** distraction is **maladaptive** when used to put off starting a solvable assignment (a controllable stressor), because the problem is left unresolved while time is lost. The strategy is therefore judged relative to the context, not in isolation.
- Q12.** **Coping flexibility** is the ability to **modify or adjust** one's coping strategies to suit the **changing demands** of a stressful situation — recognising when a strategy is not working and switching to a better one. It is associated with **better adjustment** because a person who is flexible **matches their coping to the situation**: as the demands of a stressor change, they monitor whether their strategy is helping and **switch** if it is not, rather than persisting rigidly with an ineffective response.
- Q13.** In the **short term**, an avoidance strategy can be **adaptive**: by turning attention away from the stressor it **reduces overwhelming distress** and gives the person **respite**, which is useful when nothing can be done immediately. In the **long term**, however, it becomes **maladaptive** because the stressor is **never dealt with**: if the stressor is controllable, the problem **persists or worsens**, opportunities to act are missed, and avoidance can create **further problems** (e.g. procrastination leaving less time, or substance use causing its own harm). So the strategy trades short-term relief for long-term cost.
- Q14.** Noah is showing **low coping flexibility** — he uses the **same** strategy (gaming) for every stressor, regardless of whether it fits. For a stressor that gaming does not resolve (such as a deadline), persisting rigidly with this strategy means the **stressor remains unaddressed** while time passes, so his **stress builds and his adjustment worsens**.

Higher coping flexibility would let him recognise that gaming is not reducing the stress and switch to a strategy that does.

Q15. Leila's friend is judging the strategy without considering the **context**. The death of a pet is an **uncontrollable** stressor, and in the **first few days** the grief is acute. Because **the loss itself cannot be undone**, no strategy can remove this stressor, so keeping busy is not leaving a solvable problem unattended; **distraction eases distress she has no way of acting on** and gives her respite while the acute grief settles. Given the **context-specific effectiveness** of coping strategies, her short-term use of distraction for an uncontrollable, acute stressor is **appropriate**, even though relying on avoidance indefinitely would not be. (Approach strategies remain open to Leila — talking about the loss with family, for example, engages with the stressor and is valuable — but they work on how the loss is affecting her rather than reversing it, so choosing distraction for a few days does not make her strategy “wrong”.)

Q16. (a) The **independent variable** is the students' **coping style** (predominantly approach versus predominantly avoidance); the **dependent variable** is the **psychological wellbeing score** (out of 60). (b) The **population** is **first-year university students** (at the university studied), from which the 80 participants were sampled. (c) *Directional hypothesis*: “It was hypothesised that first-year university students who are predominantly **approach** copers would report a **higher** mean psychological wellbeing score than those who are predominantly **avoidance** copers.” (d) The data **support** this: approach copers had a **higher** mean wellbeing score (**44.6**) than avoidance copers (**31.8**) — a difference of 12.8 — so more approach-oriented coping was associated with greater wellbeing. (e) A suitable ethical consideration is **confidentiality** of the participants' wellbeing results (or **informed consent**, or providing **support/referral information** after a potentially sensitive wellbeing questionnaire). (*Note: because coping style was measured, not manipulated, and participants were not randomly allocated, the study can show only an association — it cannot conclude that coping style causes the difference in wellbeing.*)

Q17. (a) The **independent variable** is the **level of coping flexibility** (high versus low); the **dependent variable** is the employees' **self-reported stress rating** (0–10). (b) Because participants were **not randomly allocated** to the high- and low-flexibility groups, the two groups may **differ on other participant variables** (for example, workload, personality or support) that could explain the difference in stress. Without control of these variables, the researchers **cannot conclude that coping flexibility caused the lower stress** — they can only report an association. (c) *Directional hypothesis*: “It was hypothesised that employees with **high** coping flexibility would report a **lower** mean stress rating during the project than employees with **low** coping flexibility.” (d) High coping flexibility might reduce stress because, as the demands of the project **change over the three weeks**, flexible employees can **monitor whether their strategy is working and switch** to a more suitable one, so their coping stays matched to the situation rather than becoming ineffective.

Q18. Two suitable **approach** strategies are: (1) **making a plan/timetable** that breaks the workload into manageable steps and scheduling time for each subject; and (2) **seeking help or information** — for example, asking the teacher to clarify a task or asking a classmate to study together. These engage directly with the workload rather than avoiding it. **Coping flexibility** would help because the workload's demands **change over time**: the student can **monitor whether a strategy is reducing the pressure** and, if one is not working (e.g. the timetable is unrealistic), **adjust or switch** to another approach strategy, keeping their coping matched to the changing demands rather than returning to avoidance.

Q19. *Model response (6 marks).* The injury sets Jordan two very different demands: a rehabilitation program he can work at, and a season he cannot get back. An **approach** strategy would have him **engage with the stressor directly** — doing the exercises his physiotherapist sets, asking how long each stage of the recovery should take, and setting small targets he can measure himself against. The rehabilitation is the **controllable** side of the situation, so an approach strategy is **well matched to that context**: the effort feeds straight back into how well the ligament heals, which is why approach coping is generally the stronger choice across months rather than days. An **avoidance** strategy — refusing to think about the injury, or staying away from the club so he does not have to watch the team play — may **reduce his distress for a while**, and in the first shocked days, before the rehabilitation program begins, that respite costs him nothing. If Jordan **keeps** avoiding, however, the exercises go undone, the recovery stalls and he loses his contact with the team as well, so the same strategy turns **maladaptive**. This is the **context-specific effectiveness** of coping: distraction that is harmless in the first week is harmful once there is a program to follow, and no amount of approach coping can restore the season — only the fitness he is able to rebuild. **Coping flexibility** joins the two ideas together. Jordan should **check whether the strategy he is using is actually easing the stress and change it as the demands change** — dropping distraction once rehabilitation begins, and, if counting daily progress starts to frustrate him when recovery slows, moving his attention to longer-term goals or to helping the team from the sidelines.

Because he **fits his coping to what each stage of the recovery asks of him** instead of repeating one response, his adjustment and wellbeing over the four months are likely to be better. *(Full marks require all three constructs applied to Jordan: at least one approach and one avoidance strategy, correctly classified; an explicit judgement of when each suits this situation, tied to what he can and cannot control and to the time frame; and coping flexibility defined and shown at work as the recovery changes.)*

Topic 2 Test A — Multiple choice

Reading time: 5 minutes. Writing time: 25 minutes. 20 questions, 20 marks. No calculator, no notes. Choose the response that is correct or that best answers the question. A correct answer scores 1 mark; an incorrect answer scores 0. Marks are not deducted for incorrect answers.

Question 1

Which of the following is a function of the central nervous system rather than of the peripheral nervous system?

- A. processing incoming sensory information and initiating a response to it
- B. carrying sensory messages from the skin and muscles towards the spinal cord
- C. carrying motor commands from the spinal cord out to the skeletal muscles
- D. carrying messages that adjust the activity of the internal organs and glands

Question 2

A netballer deliberately catches a pass. Which subdivision of the peripheral nervous system carries this motor command to the skeletal muscles of her arms and hands?

- A. the autonomic nervous system
- B. the sympathetic nervous system
- C. the parasympathetic nervous system
- D. the somatic nervous system

Question 3

Which of the following lists the structures of a spinal reflex arc in the correct order?

- A. receptor → sensory neuron → interneuron → motor neuron → effector
- B. receptor → motor neuron → interneuron → sensory neuron → effector
- C. receptor → sensory neuron → motor neuron → interneuron → effector
- D. effector → motor neuron → interneuron → sensory neuron → receptor

Question 4

A student's hand jerks away from a hot oven tray before she feels any pain. This response is best classified as:

- A. a conscious response, because she becomes aware of the heat
- B. an unconscious response, coordinated by the autonomic nervous system
- C. an unconscious response, coordinated by the spinal cord
- D. a conscious response, coordinated by the brain

Question 5

GABA binds to receptors on a post-synaptic neuron. The most likely effect is that the neuron becomes:

- A. more likely to fire an action potential, because GABA is the brain's main excitatory neurotransmitter
- B. less likely to fire an action potential, because GABA is the brain's main inhibitory neurotransmitter
- C. less likely to fire an action potential, because GABA physically blocks the synaptic gap
- D. unaffected, because GABA acts only on the neuron that released it

Question 6

Compared with a neurotransmitter such as glutamate, a neuromodulator such as dopamine typically:

- A. acts more broadly and more slowly, adjusting the activity of many neurons
- B. produces a single, fast excitatory effect on one post-synaptic neuron
- C. binds to receptors within only one synapse and is then immediately cleared
- D. always makes the post-synaptic neuron more likely to fire

Question 7

In a laboratory study, a pathway of neurons is stimulated repeatedly at high frequency. For weeks afterwards, the post-synaptic neurons in that pathway fire more readily in response to the same input. This change is best explained by:

- A. long-term depression, because repeated stimulation of a pathway makes its neurons less responsive
- B. pruning, because the pathway's unused connections have been removed
- C. long-term potentiation, because repeated, high-frequency stimulation strengthens synaptic connections
- D. the action of a neuromodulator, which briefly makes a group of neurons easier to excite

Question 8

As a violinist practises a difficult passage over many weeks, neurons in the pathway that controls the finger movements grow additional branches from their axon terminals, creating extra synaptic connections. This structural change is best described as:

- A. pruning
- B. sprouting
- C. rerouting
- D. long-term depression

Question 9

Before an exam, Ravi keeps thinking, "I'm going to fail and let everyone down." This thought is best classified as:

- A. an external stressor, because the exam is set by the school
- B. a physiological stress response, because it raises his heart rate
- C. a benign-positive appraisal, because it motivates him
- D. an internal stressor, because it arises from his own thoughts

Question 10

During a stressful week, Mia notices her heart racing and her muscles feeling tense. These reactions are best classified as:

- A. psychological stress responses
- B. physiological stress responses
- C. external stressors
- D. secondary appraisals

Question 11

A hiker suddenly sees a snake and reacts within a second, heart pounding. The pathway responsible for this rapid acute stress response is the:

- A. sympathetic nervous system stimulating the adrenal medulla to release adrenaline and noradrenaline
- B. HPA axis stimulating the adrenal cortex to release cortisol
- C. parasympathetic nervous system stimulating the adrenal cortex to release adrenaline
- D. vagus nerve stimulating the adrenal medulla to release cortisol

Question 12

After several months of financial hardship, which of the following correctly describes the biological pathway of the resulting chronic stress response?

- A. sympathetic nervous system → adrenal medulla → cortisol
- B. HPA axis → adrenal medulla → adrenaline
- C. HPA axis → adrenal cortex → cortisol
- D. HPA axis → adrenal cortex → adrenaline

Question 13

In the fight–flight–freeze response, the freeze response is best described as:

- A. a complete failure of the nervous system to respond to the threat
- B. the release of cortisol from the adrenal cortex to sustain long-term arousal
- C. a calm, relaxed state produced once the threat has fully passed
- D. an active, alert immobility, usually described as parasympathetic-dominated

Question 14

Which of the following best shows that communication in the gut–brain axis is bidirectional?

- A. the gut contains a large community of microorganisms known as the gut microbiota
- B. stress can alter activity in the gut, and signals from the gut can in turn influence mood
- C. the brain sends signals along the vagus nerve that slow digestion during a threat
- D. cortisol released during chronic stress can change the balance of the gut microbiota

Question 15

A study finds that adults with a more diverse gut microbiota report lower stress. A psychologist would correctly conclude that this finding:

- A. proves that improving the gut microbiota reduces stress
- B. shows that stress has no biological basis
- C. shows only an association, because correlational data cannot establish that the microbiota causes the lower stress
- D. cannot be valid because the gut and brain are not connected

Question 16

According to Selye's General Adaptation Syndrome, during the shock sub-phase of the alarm reaction, the body's resistance to the stressor:

- A. rises above its normal level as the sympathetic nervous system mobilises the body
- B. drops below its normal level as the body reacts as though it has been injured
- C. is sustained above normal by the ongoing release of cortisol
- D. stays exactly at its normal level

Question 17

Selye described the General Adaptation Syndrome as "non-specific". This means that:

- A. the response occurs only for specific, physical stressors
- B. the response depends on how the person appraises the stressor
- C. the pattern of physiological changes differs for each type of stressor
- D. the same general pattern of physiological changes occurs regardless of the type of stressor

Question 18

Worrying that she might fail an upcoming driving test, Sana appraises the test as a source of possible future harm. In Lazarus and Folkman's Transactional Model, this is a primary appraisal of the event as a:

- A. harm/loss
- B. benign-positive event
- C. threat
- D. challenge

Question 19

In Lazarus and Folkman's Transactional Model, secondary appraisal involves:

- A. judging whether the event is irrelevant, benign-positive or stressful
- B. evaluating one's coping resources and options for dealing with the situation
- C. a third and final stage in which a coping strategy is automatically selected
- D. the body's automatic release of stress hormones

Question 20

The night before receiving a medical result he cannot change, Tom distracts himself by watching films. Considering the context-specific effectiveness of coping, this avoidance strategy is best described as:

- A. maladaptive, because avoidance strategies are always harmful
- B. an approach strategy, because he is actively choosing an activity
- C. an example of high coping flexibility, because he is switching strategies
- D. adaptive, because the stressor is uncontrollable and immediate, so distraction eases distress he cannot act on

End of Topic 2 Test A

Test A — solutions

Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	A	6	A	11	A	16	B
2	D	7	C	12	C	17	D
3	A	8	B	13	D	18	C
4	C	9	D	14	B	19	B
5	B	10	B	15	C	20	D

Q1. A — the CNS (the brain and the spinal cord) receives, processes and integrates information and initiates a response to it; carrying messages towards the CNS (B), out to the skeletal muscles (C) and to the internal organs and glands (D) is the work of the peripheral nervous system. **Q2.** D — deliberate, voluntary movement of skeletal muscle is carried by the somatic nervous system; the autonomic, sympathetic and parasympathetic divisions control involuntary internal organs. **Q3.** A — the reflex arc runs receptor → sensory (afferent) neuron → interneuron → motor (efferent) neuron → effector; the other orders misplace the neurons. **Q4.** C — a hand-withdrawal reflex is unconscious and is coordinated by the spinal cord, not by the brain (D) and not by the autonomic nervous system (B); awareness of the heat arrives only afterwards (A). **Q5.** B — GABA is the brain's main inhibitory neurotransmitter, so binding to receptors on the post-synaptic neuron decreases the likelihood that it will fire an action potential. It is not excitatory (A), it binds to specific receptor sites rather than blocking the synaptic gap (C), and its effect is on the receiving neuron, not on the neuron that released it (D). **Q6.** A — a neuromodulator acts broadly and slowly, adjusting the activity of many neurons at once; the fast, local action on a single synapse described in B and C is that of a neurotransmitter, and D is wrong because a neuromodulator can make firing either more or less likely. **Q7.** C — a lasting increase in how readily the post-synaptic neurons fire after repeated, high-frequency stimulation is long-term potentiation. Long-term depression (A) is a weakening, produced by repeated low-frequency stimulation; pruning (B) removes connections rather than strengthening them; and a neuromodulator (D) would produce a broad, temporary change in excitability, not one lasting weeks. **Q8.** B — growing new branches from the axon terminals to create additional synaptic connections is sprouting; pruning removes connections, rerouting forms an alternative route around a lost or damaged one, and LTD is a functional weakening rather than a structural growth. **Q9.** D — a self-generated worrying thought is an internal stressor; the exam itself would be the external stressor. **Q10.** B — a racing heart and muscle tension are bodily (physiological) responses, not cognitive or emotional (psychological) ones. **Q11.** A — a sudden threat triggers the fast sympathetic → adrenal medulla → adrenaline/noradrenaline pathway; the HPA-axis/cortisol pathway (B) is the slow, chronic response. **Q12.** C — chronic stress runs through the HPA axis and the adrenal cortex, which releases cortisol; the medulla and adrenaline belong to the acute response. **Q13.** D — freeze is an active, alert immobility usually described as parasympathetic-dominated, not a failure to respond (A), the chronic cortisol response (B), or a post-threat calm (C). **Q14.** B — bidirectional means signals travel in both directions, and only B pairs a brain-to-gut effect with a gut-to-brain effect; C and D are both true statements but describe top-down (brain-to-gut) communication only, and A describes the microbiota rather than the axis. **Q15.** C — the data are correlational, so they show only an association; causation and its direction cannot be established from a correlation. **Q16.** B — in shock the body reacts as if injured and resistance drops below normal; it rises above normal only in counter-shock (A) and is cortisol-sustained in the resistance stage (C). **Q17.** D — “non-specific” means the same general physiological pattern occurs regardless of the type of stressor. **Q18.** C — anticipated future harm that has not yet occurred is a threat appraisal; harm/loss (A) is damage already done and challenge (D) is potential gain. **Q19.** B — secondary appraisal evaluates coping resources and options; A is primary appraisal, and there is no automatic third “strategy-selection” stage (C). **Q20.** D — for an uncontrollable, immediate stressor, distraction eases distress that cannot be acted on, so it is contextually adaptive; avoidance is not “always” harmful (A).

Topic 2 Test B — Short answer

Writing time: 35 minutes. Total 30 marks. No calculator, no notes. Answer all questions. Where more than one mark is available, full marks require the depth the command term and mark allocation ask for.

Question 1 (4 marks)

Jesse steps on a piece of broken glass and instantly pulls his foot away, feeling the pain a moment later.

- a. Name the type of response Jesse's foot withdrawal is, and name the structure of the central nervous system that coordinates it. 2 marks
- b. Later that evening Jesse sees a second piece of glass on the floor and deliberately steps around it. Explain how this response differs from his earlier foot withdrawal, referring to awareness, voluntary control and the part of the nervous system that coordinates each. 2 marks

Question 2 (4 marks)

- a. Distinguish between an excitatory and an inhibitory neurotransmitter, naming an example of each. 2 marks
- b. Explain how a neuromodulator differs from a neurotransmitter in the way it acts. 2 marks

Question 3 (4 marks)

- a. A student writes: "Long-term depression occurs when a neural pathway receives no stimulation at all." Identify the error in this statement and correct it. 2 marks
- b. Distinguish between rerouting and sprouting as structural changes to the connections between neurons. 2 marks

Question 4 (6 marks)

A psychologist investigates whether spending time with a therapy dog lowers the physiological arousal of students during exam week. Eighty first-year university students are randomly allocated to a **therapy dog** group, who spend 20 minutes in a room with a therapy dog and its handler, or a **control** group, who spend 20 minutes reading quietly in the same room. Each student's systolic blood pressure is measured immediately afterwards. The mean results are shown below.

Group	Mean systolic blood pressure (mmHg)	Standard deviation
Therapy dog	118.4	8.6
Control	124.7	9.1

- a. Identify the independent variable and the dependent variable. 2 marks
- b. Write a directional (one-tailed) hypothesis for this study that names both levels of the independent variable and a directional dependent variable. 2 marks
- c. The therapy dog group recorded the lower mean systolic blood pressure. Identify one extraneous variable that could offer an alternative explanation for this difference, and explain how randomly allocating the students to the two groups helps to control it. 2 marks

Question 5 (3 marks)

- a. Distinguish between the shock and counter-shock sub-phases of the alarm reaction in terms of what happens to the body's resistance to the stressor. 2 marks
- b. Name the division of the nervous system responsible for the arousal during counter-shock. 1 mark

Question 6 (3 marks)

Distinguish between the acute stress response and the chronic stress response. In your answer, refer to the pathway (nervous system or axis), the part of the adrenal gland, and the main hormone(s) involved in each.

Question 7 (6 marks)

Priya has been told that the café where she works will close in three months, so she will lose her job. At first she cannot stop worrying and appraises the news as a serious threat. Using Lazarus and Folkman's Transactional Model, explain how Priya's appraisal of this stressor influences whether she experiences stress. Then, referring to approach and avoidance coping strategies, the context-specific effectiveness of a strategy, and coping flexibility, explain how Priya might cope with this stressor over the coming months.

End of Topic 2 Test B

Test B — solutions

Q1. (a) Jesse's foot withdrawal is a **spinal reflex** (an unconscious response); it is coordinated by the **spinal cord**, not the brain. (b) Stepping around the second piece of glass is a **conscious** response: Jesse is **aware** of the glass before he moves, the movement is **voluntary** and he could choose not to make it, and it is initiated by the **brain**, which plans the movement before the command is sent to his leg muscles. His foot withdrawal was **unconscious**: it happened **automatically, without awareness or intention**, and it was coordinated by the **spinal cord**, with the message reaching the brain — and the pain being felt — only after the foot had already moved.

Q2. (a) An **excitatory** neurotransmitter **increases the likelihood** that the post-synaptic neuron will fire an action potential — for example **glutamate**. An **inhibitory** neurotransmitter **decreases the likelihood** that it will fire — for example **GABA**. (b) A **neurotransmitter** acts **locally and quickly**, producing a **direct excitatory or inhibitory** effect on **one** post-synaptic neuron. A **neuromodulator** (such as dopamine or serotonin) acts **more broadly and more slowly, modulating the activity of many neurons at once** and producing a **range of effects** on brain activity rather than a single fast signal.

Q3. (a) The **error** is the claim that LTD follows an **absence** of stimulation. The **correction** is that long-term depression is produced by **repeated low-frequency (weak, low-level) stimulation** of the synapse: the pathway is still being activated, just weakly and infrequently, and it is that low-level activation that actively weakens the connection. (b) **Rerouting** forms a **new or alternative connection** between neurons — a different route to a target neuron, typically when an existing route has been lost or damaged; **sprouting** grows **new branches** from a neuron's axon terminals or dendrites, adding connections along a pathway that is already in use. Rerouting therefore changes **where** the signal travels, whereas sprouting increases **how many** connections an existing pathway has.

Q4. (a) The **independent variable** is **how each student spends the 20 minutes** — with the therapy dog or reading quietly. The **dependent variable** is the student's **systolic blood pressure (in mmHg)** measured immediately afterwards. (b) A suitable **directional hypothesis** is: *"It was hypothesised that first-year university students who spend 20 minutes with a therapy dog during exam week will have a lower mean systolic blood pressure than students who spend 20 minutes reading quietly."* This names **both levels** of the IV (therapy dog vs quiet reading) and gives the DV a **direction** (a *lower* blood pressure). (c) One **extraneous variable** is **how stressed each student already was** — for example, how many exams they still had to sit. (Other acceptable answers: fitness, caffeine consumed that morning, or how much a student likes or fears dogs.) If more of the already-calm students happened to be placed in the therapy dog group, their **lower mean blood pressure** could be explained by that difference rather than by the dog. **Random allocation** gives every student an **equal chance** of being placed in either group, so such participant differences are **spread evenly across the two groups** instead of building up in one of them; the groups therefore begin **similar on average**, and the difference between the group means is more likely to be due to the IV.

Q5. (a) The two sub-phases move the body's resistance in **opposite** directions. In **shock**, the body has not yet mobilised its defences and briefly responds as though it had been **injured**, so its resistance to the stressor sits **below its normal level**. In **counter-shock**, stress hormones are released and the body mobilises for action, lifting its resistance to the stressor **above its normal level**. (b) The **sympathetic nervous system** produces the arousal during counter-shock.

Q6. In the **acute** stress response, a sudden stressor activates the **sympathetic nervous system**, which stimulates the **adrenal medulla** (the **inner** part of the adrenal gland) to release **adrenaline** (and noradrenaline); this neural pathway is **fast** and the arousal is **brief**. In the **chronic** stress response, a prolonged stressor activates the **HPA axis**, and the **adrenal cortex** (the **outer** part of the adrenal gland) releases **cortisol**; this hormonal pathway is **slower** but keeps the body mobilised in a **sustained** way. The two therefore differ in the pathway (sympathetic nervous system vs HPA axis), the adrenal structure (medulla vs cortex) and the hormone (adrenaline/noradrenaline vs cortisol). (*Full marks require all three points of contrast.*)

Q7. Model response (6 marks). According to the Transactional Model, whether Priya feels stress depends not on the news itself but on how she **appraises** it. In her **primary appraisal**, Priya judges what the news means for her wellbeing: she appraises it as **stressful**, and specifically a **threat**, because the job loss represents **potential future harm** that has not yet happened. In her **secondary appraisal**, she evaluates her **coping resources and options** — her savings, her skills, the time she has to find another job, and the support available to her. The model holds that Priya will experience stress if she appraises the **demands** of the situation as **exceeding** her **coping resources**, and little

stress if she appraises her resources as **sufficient**; it is her **subjective appraisal**, not the event alone, that determines her stress.

Priya can then cope using different strategies. An **approach** strategy would have her **engage with the stressor directly** — updating her résumé, searching for jobs and seeking advice. Because the job loss is **at least partly controllable** (she can prepare for it and look for other work), an approach strategy is **well-suited to this context** and is likely to be **adaptive in the long term**, since it acts on the demands. An **avoidance** strategy — not thinking about it, or distracting herself — might **ease her distress in the short term**, which can be reasonable **early on**; but if she **relies** on avoidance over the three months it becomes **maladaptive**, because the problem is left unaddressed and the chance to prepare is missed. This illustrates the **context-specific effectiveness** of coping: a strategy's value depends on the situation, including the stressor's **controllability** and **time frame**. Finally, **copng flexibility** — **monitoring** whether her current strategy is working and **switching** to a more suitable one as the situation changes (for example, moving from distraction to active job-seeking as the closing date nears) — would help Priya **match her coping to the changing demands**, which is associated with **better adjustment**. *(Full marks require: a correct primary appraisal with its type (threat) and reference to secondary appraisal/coping resources; correct use of approach and avoidance strategies; a judgement of context-specific effectiveness tied to controllability or time frame; and a correct application of coping flexibility.)*